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THE PLANT DISEASE REPORTER

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THE PLANT DISEASE SURVEY

Division of Mycology and Disease Survey
BUREAU OF PLANT INDUSTRY, SOILS, AND AGRICULTURAL ENGINEERING
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The Plant Disease Reporter is issued as a service to plant pathologists throughout the United States. It contains reports, summaries, observations, and comments submitted voluntarily by qualified observers. These reports often are in the form of suggestions, queries, and opinions, frequently purely tentative, offered for consideration or discussion rather than as matters of established fact. In accepting and publishing this material the Division of Mycology and Disease Survey serves merely as an informational clearing house. It does not assume responsibility for the subject matter.

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THE PLANT DISEASE SURVEY
DIVISION OF MYCOLOGY AND DISEASE SURVEY

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AN EASTERN OREGON FRAGARIA VESCA SUITABLE AS AN INDICATOR FOR SOME STRAWBERRY VIRUS DISEASES

P. W. Miller

As has been pointed out by Plakidas (4), Zeller (6), Harris (2), and others, the cultivated varieties of strawberries differ greatly in their sensitiveness to virus diseases. Some varieties such as the Rockhill and Huxley show little, if any, external symptoms of disease though they are known to be carriers of viruses. Moreover, during mid-summer even the more susceptible varieties often show little evidence of disease. However, if the causal virus is transmitted either by stolon-grafting or by aphids to a more sensitive indicator plant, that is, one that is easily infected and readily shows disease symptoms, the presence of a virus disease can be demonstrated.

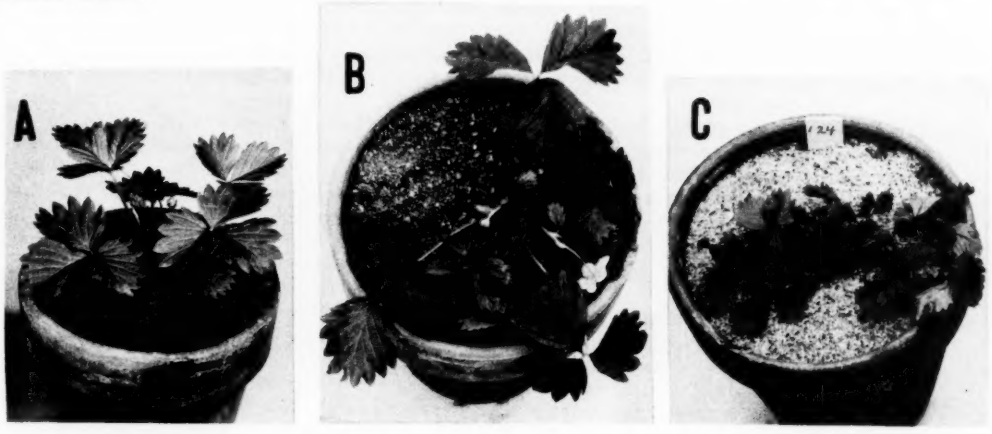

FIGURE 1. Fragaria vesca from Seneca, Oregon: (A) Healthy plant. (B) Plant infected with yellows induced by inoculation with viruliferous aphids; photographed 22 days after inoculation, (C) Plant infected with yellows induced by inoculation with viruliferous aphids; photographed 36 days after inoculation.

In England, Harris and King (3) have shown that the wild woodland strawberry (Fragaria vesca L.) and certain strains of F. virginiana Duchesne are good indicator plants, being very sensitive and showing symptoms at all times. In the United States Demaree (1) has also found F. vesca received from Harris a good indicator. Thomas (5) has shown that a strain of F. vesca called var. californica is also a good indicator plant.

The chief objection to the use of some strains of F. vesca is that the stolons are of small diameter, which makes stolon-grafting difficult. Moreover, some of the strains of F. vesca are not especially good indicator plants.

During the course of the writer's investigations on the virus diseases of strawberry, collections were made of a number of native Oregon wild F. vesca variants and they were tested for sensitiveness to yellows and crinkle. One of these collected by R. G. Rosenstiel in eastern Oregon near Seneca proved to be very sensitive to these diseases and, in addition, had runners of relatively large diameters (Figure 1, A). Primary stolons of this selection from relatively small plants measure 1 to 2 mm. in diameter, averaging about 1.5 mm.

In aphid transmission studies, inoculated vigorous plants of this F. vesca, incubated at a temperature of 65° to 70° F., showed visible symptoms of yellows in ten days to two weeks after viruliferous aphids from infected plants has been caged thereon (Figure 1, B). In a month to six weeks after inoculation, the plants were usually dead or almost so (Figure 1, C).

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BUREAU OF PLANT INDUSTRY, SOILS AND AGRICULTURAL ENGINEERING, OREGON STATE COLLEGE, CORVALLIS

SUPPRESSION OF STRAWBERRY YELLOWS BY CONTROLLING THE APHID VECTOR CAPITOPHORUS FRAGAEFOLII (Ckll.)[1]

E. P. Breakey[2] and Leo Campbell[3]

For many years, the yellows disease has been a constant threat to the strawberry industry of the Pacific Northwest. This is because commercial plantings in the area have been almost entirely of the highly susceptible Marshall variety. The acreage planted to strawberries in western Washington has been greatly expanded in recent years, particularly around Lynden in Whatcom County. This development has been followed by a marked increase in the yellows disease with attendant decrease in yields of as much as 75 percent.

Initial efforts to control the disease consisted primarily in the production and use of certified planting stock, i.e., stock that had been certified by the Washington State Department of Agriculture as containing less than 1 percent infected plants. This was supplemented by roguing diseased plants as they appeared in new plantings. Such control measures were ineffective, however, unless the planting was well isolated from infected mature fields. Even then the results were often discouraging, since the virus spread from diseased plants to healthy plants faster than the symptoms developed.

Plakidas (1) and Massee (2) have shown that the yellows disease is due to virus spread by the strawberry aphid. While Prentice and Harris (3 and 4) have answered many questions regarding the virus complex known as the yellows disease, and have helped prove beyond question that the aphid is the vector, no one has been able to recommend a practical solution to the problem. Plant breeders have attempted to develop acceptable varieties that would be either resistant to the disease or tolerant of it (7 and 8). Experience has shown, however, that this approach will probably take many years to complete and that the trade may be reluctant to accept the hybrids offered.

The development of highly effective new insecticides suggested that it might be possible to control the aphid and thus slow down or stop the spread of the virus. It was thought that if new plantings of certified stock could be dusted at regular intervals with insecticides, it might enable the growers to rogue effectively and to again produce the Marshall strawberry profitably.

Screening tests in 1947 showed that some of the new insecticides were not effective against the strawberry aphid. Dusts containing 5 percent DDT were only partially effective. Moreover, DDT in amounts as low as five pounds per acre retarded the development of the strawberry plants and seemed to aggravate the spider mite problem. BHC dusts (0.5 to 1% gamma isomer of benzene hexachloride) proved to be more effective against aphids and non-toxic to plants.

The objective of the expanded work in 1948 was to protect new plantings so the grower could rogue effectively. Some new insecticides, particularly parathion, were credited with a residual effect extending over a considerable period of time. Tests were set up to determine if periodic application of the new insecticides would protect new plantings so that, by carefully roguing all plants showing symptoms of the disease, the spread of the virus could be checked. Those insecticides that had been the most promising in screening tests made during the previous year were again tested along with certain others that had been suggested.

MATERIALS AND METHODS - 1948

The following insecticide dusts were tested as controls for the strawberry aphid: Parathion (0.5% 0-0-diethyl 0-p-nitrophenyl thiophosphate); BHC (0.5% gamma isomer, benzene hexachloride); Rhothane (DDD) (5% dichlorodiphenyl dichloroethane); and methoxychlor (5% bis (methoxyphenyl) trichloroethane).

A planting of Marshall strawberries was established near Lynden for the purpose of testing the effectiveness of the new insecticides. Four insecticides were tested against an untreated check, using nine 100-foot rows per replicate and five replicates per treatment. The replicates were randomized to compensate for any variation, particularly in population, that might exist. Data were taken from the center three rows of each replicate, leaving three rows on either side for buffers.

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[1]Scientific Paper No. 984, Washington Agricultural Experiment Stations, Institute of Agricultural Sciences, State College of Washington, Pullman. These studies were assisted by grants in aid for research by the Washington State Department of Agriculture and the American Cyanamid Company.
[2]Associate Entomologist, Western Washington Experiment Station, Puyallup,
[3]Assistant Plant Pathologist, Western Washington Experiment Station, Puyallup.

The planting was directly south of a mature field, the two being separated by a turning area that was kept clean of vegetation. A random sampling of plants in the mature field disclosed that approximately 35 percent were diseased.

Five applications were made at intervals of two weeks, beginning one week after planting, using 8 to 10 pounds of dust per acre per application. The dusting program was terminated on July 10, soon after current infection had developed to a point where diseased plants could readily be identified. This was soon after the berry harvest had been completed. All applications were made with a power duster mounted on a small tractor and adapted to treating three rows at a time.

Experimental Results

Data on the effectiveness of the insecticides as controls for the strawberry aphid are summarized in Table 1. Counts were made on July 8 and 9, soon after the berry harvest had been completed. Aphid counts were obtained by picking, at random, twenty tri-foliate leaves of medium maturity from the center three rows of each replicate. Since there were five replicates the living aphids on one-hundred leaves from each treatment were counted.

Table 1. Data on the control of the strawberry aphid - 1948. Living aphids per 20 tri-foliate leaves.

Replicate	Parathion	BHC	Rhothane	Methoxychlor	Check
1	0	4	74	477	173
2	2	43	140	328	184
3	20	23	60	315	121
4	0	16	72	261	94
5	10	5	155	152	120
Total	32	91	501	1533	692
Average per 3-foliate leaf	.32	.91	5.01	15.33	6.92

Of the four insecticides tested, parathion and benzene hexachloride were the most effective. Methoxychlor actually seemed to benefit or promote the development of the aphids. Plots treated with this insecticide contained from two to three times as many as the untreated checks. Plots dusted with the Rhothane and methoxychlor were more heavily infested with spider mites than were the untreated checks. Parathion gave excellent control of the aphids as well as being outstanding in the control of the spider mites.

Counts to determine the incidence of yellows plants were made on July 27. These data are summarized in Table 2. Counts were delayed to allow ample time for the development of symptoms in currently infected plants.

Benzene hexachloride gave nearly 4 percent depression in the spread of yellows, and parathion over 12 percent, compared with the untreated checks. The depression in the spread of yellows where benzene hexachloride and parathion were used does not seem impressive until the proximity of the mature field, which served as a constant source of viruliferous aphids, is considered. Data from the plots, however, indicate that the spread of yellows into new plantings cannot be satisfactorily prevented if aphids are left uncontrolled in nearby mature fields infected with the disease.

Seasonal Activity of the Aphids

Many migratory alatae were produced during the fruiting season of the strawberry. The first of these appeared soon after the blossoms began to open. Winged aphids were present in numbers on May 17. On May 29, they were noted flying in great numbers from infected mature fields to newly planted fields. About six weeks later, July 9, yellows disease showed up all over the new plantings. Apparently it required about six weeks for the virus to produce symptoms. The infection was as high as 70 percent in many new plantings in the area as a result of the distribution of the virus by these migratory aphids.

Table 2. Data on the depression of the spread of yellows by the use of insecticides to control the strawberry aphid - 1948.

Replicates	Total number of plants and number of yellows-free plants (healthy) in each of five replicates of four insecticides and untreated checks.									
	Parathion		B.H.C.		Rhothane		Methoxychlor		Checks	
	Total no. plants	Healthy plants	Total no. plants	Healthy plants	Total no. plants	Healthy plants	Total no. plants	Healthy plants	Total no. plants	Healthy plants
1	226	24	220	10	214	5	249	16	235	14
2	237	67	249	24	253	14	244	8	249	16
3	230	38	245	48	261	23	261	12	238	40
4	256	44	262	32	251	6	264	6	263	15
5	262	20	282	12	276	10	256	10	271	10
Total	1211	193	1258	126	1255	58	1274	52	1256	95
%Healthy[1] plants	24.50		15.40		7.11		6.27		11.63	
% D.Y.[2]	+12.87		+ 3.77		- 4.52		- 5.36			

[1]Corrected for original percentage of yellows.
[2]D.Y. - percent depression of yellows per treatment.

While considerably less virus showed up in some of the treated plots than appeared in the untreated checks, no treatment gave adequate control. Alatae were found that had survived long enough to insert their mouth parts into the strawberry plants, even though the residual action of the insecticides had killed them soon after beginning to feed. In several instances, alatae were found dead and still attached to the plants by their mouth parts. Some of these had given birth to young. The young aphids apparently had been killed by the residue of the insecticides and some remained held in place by the plant hairs.

It became increasingly evident that protection of the new planting alone was insufficient, even though the insecticides gave good control of the aphids. Too many alatae lived long enough to transmit the disease to new plants even though they were killed soon after arriving.

When attempting to analyze the situation with reference to all possible factors, the question arose as to what stimulus or stimuli could cause the production of migratory alatae. Of those suggested, crowding seemed to be the most logical, if allowance were made for the possibility that numbers which might produce the effect of crowding on one species might not apply to others. An infestation of the strawberry aphid never builds up in numbers comparable to those attained by such species as the bean aphid or the cabbage aphid. If crowding were responsible for the development of these winged migrants, then if the population were to be greatly reduced just before the initiation of this migratory period, the stimulus of crowding would be removed and few, if any, alatae would develop.

An observation reported by Greenslade (5) encouraged us to test the validity of this theory. This worker noted that the population of the European strawberry aphid C. fragariae (6) was so reduced by the severe winter of 1939-40 that normal numbers were not reached until September and no alatae were found in the spring when a large proportion of the aphids normally develop wings.

MATERIALS AND METHODS - 1949

Plots were established on an isolated farm west of Lynden where a new planting adjoined a mature field. This location was selected to determine if the aphid population in the mature planting could be sufficiently reduced so that the new planting could be protected by periodical dusting.

The mature planting consisted of about four acres and was separated from the new planting by a driveway. It was so heavily infected with yellows disease that the grower had seriously considered plowing it under in the late summer of 1948. The new planting consisted of 120 rows ranging in length from 250-400 feet. This made it possible to again use nine rows in each replicate and to replicate each treatment four times. Rows not needed for the plots were dusted

periodically with parathion. Data were to be taken from the three center rows of each replicate. As nearly as could be determined, the stock used for the new planting contained from 10 to 15 percent yellows at the time of planting.

The mature planting was dusted on April 26 and 27 with parathion (1%) when the strawberry blossoms started to open, using about 40 pounds of dust per acre per application. The following dusts were used in the new planting within one week after planting: parathion (1%), TEPP (tetraethyl prophosphate, 1%), and BHC (benzene hexachloride, 1% gamma isomer). Both the mature planting and the new planting were again dusted two weeks later. By this time there was a considerable number of blossoms open in the mature planting so dusting was stopped to avoid danger of a poisonous residue on the fruit. Dusting was continued on the new planting, however, at intervals of two weeks up to July 21 (two weeks after the completion of harvest), using 8 to 10 pounds of dust per acre per application.

Experimental Results

An examination of the mature planting when the second application was made disclosed that the aphid population had been greatly reduced by the first application. When data were being taken near the end of harvest, June 29, the aphid population in the mature planting was still very low. Moreover, no alatae were found in this planting at any time, though they were present in numbers in other plantings in the same neighborhood where no dusts had been applied. A very few winged aphids were found in the new planting. Apparently they flew in from the surrounding territory. Some of these had already been killed by the residual action of the insecticides. Counts made in untreated mature plantings in the same general area disclosed a heavy aphid population.

Just after harvest -- the end of the aphid migratory period -- the count in the mature field revealed 40 aphids per 100 tri-foliate leaves, while in the adjoining plots the count per 100 trifoliate leaves was: benzene hexachloride 5; parathion 14; and tetraethyl pyrophosphate 16 (Table 3). A mature field a few miles away, which had not been treated, yielded 331 aphids per 100 trifoliate leaves. The same method of taking data was used as had been employed in 1947 and 1948.

Table 3. Data on the control of the strawberry aphid - 1949.
Living aphids per 25 tri-foliate leaves.

	New planting [1]		Mature planting [2]	
Replicates	BHC	Parathion	TEPP	Parathion
1	0	3	11	6
2	2	7	3	0
3	2	2	1	12
4	1	2	1	22
Total	5	14	16	40
Average per trifoliate leaf	.05	.14	.16	.40

[1] Dusted at intervals of 2 weeks beginning 1 week after setting of plants.
[2] Dusted twice before blossoming, an interval of 2 weeks between applications.

The data on the aphids and on the spread of yellows in treated plots compare very favorably. The average percentage of yellows for each material in the plots when the data were taken in September was: benzene hexachloride 9.5; parathion 13.37; and tetraethyl pyrophosphate 10.55. It will be recalled that the stock used in setting this field contained from 10 to 15 percent yellows when planted. These data have been summarized in Table 4. They show that the spread of the disease had been checked and apparently stopped. In untreated commercial fields in comparative situations, the incidence of yellows was generally around 75 percent.

These data show that the migration of aphids and the spread of yellows within a new field or from a mature field into an adjoining new field can be prevented or materially reduced.

Table 4. Data on the control of strawberry yellows by the use of insecticides for the control of the strawberry aphid in plots adjoining a treated mature field - 1949.

Replicates	Parathion		BHC		TEPP	
	Total no. plants	No. yellows plants	Total no. plants	No. yellows plants	Total no. plants	No. yellows plants
1	370	65	542	56	533	40
2	525	66	528	42	531	43
3	534	69	505	74	513	49
4	403	45	415	38	369	53
Total	1832	245	1990	210	1946	185
Percent yellows		13.37		10.55		9.50

New Plantings in Isolation Treated and Rogued

Another planting was established on a stock farm south and east of Lynden in complete isolation from other strawberry fields. These plots were treated with dusts containing BHC (benzene hexachloride, 1% gamma isomer), parathion (1%) and TEPP (tetraethyl pyrophosphate, 1%) in five replications of nine 50-foot rows each. Applications were made at intervals of two weeks throughout the growing season, using 8 to 10 pounds of dust per acre per application. The planting stock contained only a trace of yellows. Immediately after planting, every tenth plant was replaced by one having the disease. Sixty-two days after planting, all diseased plants were removed.

It was difficult to find even an occasional aphid in these plots at any time during the growing season. Not one alate was found. When the final data on the incidence of yellows were taken in September, the disease was found in correspondingly low percentages. These data are summarized in Table 5. The average percentage of yellows for each of the materials was: benzene hexachloride 1.67; parathion 0.72; and tetraethyl pyrophosphate 0.47.

Table 5. Data on the control of strawberry yellows by the use of insecticides for the control of the strawberry aphid in plots in isolation - 1949.

Replicates	Parathion		TEPP		BHC	
	Total no. plants	No. yellows plants	Total no. plants	No. yellows plants	Total no. plants	No. yellows plants
1	72	0	91	0	75	4
2	86	0	85	1	63	1
3	100	0	82	1	68	0
4	94	0	94	0	83	1
5	61	3	74	0	70	0
Total	413	3	426	2	359	6
Percent yellows		0.72		0.47		1.67

The data from these plots indicate that yellows was controlled in an isolated field by keeping the aphid population down with an effective insecticide and by careful roguing.

Seasonal Activity of Aphids

Migratory aphids appeared again in 1949, just as they did in 1948. The only difference noted was that the population apparently took longer to build up in 1949. The winter of 1948-49 was much more severe than that of 1947-48. This may have reduced the population somewhat and caused the delay in the build-up of the insect. However, it began and ended very much as it did in 1948. Apparently we can safely tie the development of the population to the development of the

strawberries or the host plant.

MATERIALS AND METHODS - 1950

Growers in the area had watched with great interest the progress of our efforts to control the yellows disease by controlling the strawberry aphid. Encouraged by the results, they organized early in 1950 to apply control measures on a county-wide basis. Meetings were held to formulate a program, and the writers were invited to report the progress of their studies and to act as advisors. A mimeographed circular of information was prepared and distributed among the growers to assist in the development of this program.

Experimental results obtained in 1949 were so promising that the tests were repeated in 1950. Materials used and the timing of the applications all appeared satisfactory in 1949. So were the locations of the plots, particularly with reference to sources of infection and distribution of the aphid population. However, the 1950 tests were a further check on the soundness of the program.

In addition to repeating the 1949 tests, four isolated plantings were established in four widely separated localities in western Washington. Each planting contained from three to six rows of yellows-free certified stock bordered on each side by four to six rows of stock containing approximately 20 percent diseased plants. One of these plantings was located on the grounds of the Northwestern Washington Experiment Station near Mount Vernon, and another at the Southwestern Washington Experiment Station near Vancouver. Three of these plots were dusted with parathion at intervals of two weeks, beginning one week after planting and continuing until the end of August. The fourth plot was maintained as an untreated check.

Experimental Results

Data on the effectiveness of the treatments were taken soon after strawberry harvest had been completed. No aphids were found in any plots that had been dusted. A very few aphids were found in the adjoining mature field. This field had been dusted in 1949 and 1950. Applications were made early in the season to avoid a poisonous residue on the fruit. No alatae were found in either the experimental plots or the mature field. There was no increase in the incidence of yellows in either the mature field or in the new planting where we had located our test plots.

When data were taken in the special plantings during the first part of September, no increase in yellows could be detected in the uncertified stock. No infected plants were found in the center rows of certified plants in the three plots which were kept dusted. In the untreated check plot, however, 38.18 percent of the certified plants showed yellows and the disease showed a corresponding increase in the uncertified stock.

A field day was held in late summer to inspect the growers' fields and to learn how effective their control program had been. About 80 percent of the growers in the area had participated. Those who had made a determined effort to use the control program as recommended were very successful, while those who tried to get by with less were only partially successful. Some were apparently under the impression that roguing would no longer be necessary. This is unfortunate, for roguing new fields is a very important part of the control program.

SUMMARY

The yellows disease of the strawberry has become the limiting factor in the production of the Marshall variety in the Pacific Northwest.

Efforts to control the disease consisted primarily in the planting of certified disease-free stock supplemented by the roguing of infected plants from new plantings.

The expansion of the industry has been accompanied by a steady increase in the incidence of the yellows disease, however. Infected plants require about six weeks to develop distinct symptoms, under field conditions. During this time, the strawberry aphid Capitophorus fragaefolii (Ckll.) spreads the disease from infective plants and nullifies, to a considerable extent, the effectiveness of roguing.

Studies initiated in 1947 show that the timely application of insecticides will control the strawberry aphid and prevent the spread of the yellows disease, if the field is isolated or protected against migratory alatae from nearby infected fields.

Applications of insecticide dusts were timed to destroy the aphid population in mature plantings, prevent the production of migratory alatae, and protect new plantings against infection.

Parathion, tetraethyl pyrophosphate, and the gamma isomer of benzene hexachloride, when

applied as 1 percent dusts, each gave adequate control of the strawberry aphid.

DDT and methoxychlor failed to control the aphids and aggravated the spider mite problem.

Migratory alatae are produced primarily in mature plantings during the fruiting season of the strawberry. Alatae began appearing soon after the strawberries started to blossom and continued to appear in increasing numbers until harvest. By the end of harvest, no alatae could be found.

Dusting mature fields early in the season, just as the strawberries started to blossom, effectively reduced the aphid population without the danger of leaving a poisonous residue on the fruit. It also prevented the development of migratory alatae.

Dusting new plantings at intervals of two weeks, beginning about one week after setting the plants and continuing until after the strawberry harvest had been completed, prevented further infection.

Roguing infected plants from new plantings as soon as the symptoms develop is an essential part of the control program. As long as such plants persist, they are hazards to the welfare of the industry.

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THE RELATIONSHIP BETWEEN ARGENTINA'S LEPRA EXPLOSIVA AND FLORIDA'S SCALY BARK, WITH IMPLICATIONS FOR THE FLORIDA CITRUS GROWER

L. C. Knorr and E. P. DuCharme [1]

The current importance of the citrus disease known as lepra explosiva in Argentina prompts an examination into the relationship between this disease and a similar one in Florida; namely, scaly bark or nailhead rust. If a comparison proves these two troubles to be the same, then the Florida citrus grower should be alerted to the destructiveness of lepra as it presently occurs in Argentina.

With the passing of tristeza -- that virus disease which has practically eliminated itself as a problem by killing off all orange, grapefruit, and mandarin trees on the susceptible sour orange rootstock - lepra is emerging from its secondary position to become the present Number One disease of Argentina's citrus industry. It is currently attacking groves not previously killed by tristeza, and areas involved are increasingly becoming more extensive.

Damage from lepra occurs in three ways. First, and most obvious, is that due to a spotting of the fruit. So much of Paraguay's sweet orange crop in the past few years has appeared as if covered with the rusty heads of 16-penny nails that its citrus export trade is finished. Citrus used to travel from this neighbor on the north down the Parana into Buenos Aires; now after lepra's attack in Paraguay, the movement is reversed, citrus going upstream to Paraguay.

Spotting also leads to a dropping of the fruit, and in badly infected groves losses from this source involve up to 75 percent of the crop.

A second type of damage is in the reduction of photosynthetic area. Severe spotting causes, in addition to a decrease in the green surface, a reduction in the number of leaves remaining on the tree.

The third kind of damage, and this more important though less obvious initially than damage to fruit or leaves, is the long-range effect on the tree itself. Bark lesions are small at first, but enlarge constantly with the tree's growth, and in time produce considerable damage. Already because of this phase some groves are out of production.

To date the literature on the relationship between lepra and Florida scaly bark is vague. Fawcett in his book "Citrus Diseases and their Control" (1936) points out that the malady called lepra explosiva in Argentina appears to have the same characteristics as scaly bark found in Florida.

To what extent, then, are these characteristics actually the same, or at least sufficiently close as to warrant believing the two diseases to be synonymous? To answer this question the authors, both currently stationed in Argentina but having previous experience with scaly bark in Florida, have drawn a point-for-point comparison between both troubles as they occur in the field. This comparison is presented here as Table I, together with comparable illustrations of the two diseases (Figures 1, 2, 3, 4).

A review of the similarities brought out in Table 1 leads to the conclusion that in their fundamental characteristics lepra explosiva and Florida scaly bark are synonymous. The following symptomatological characterization applies to both conditions: a disease affecting bark, leaves, and fruit, primarily of sweet and sour orange; on young bark, lesions appearing as small, round, yellow spots, later becoming chestnut-brown, elevated, glazed, brittle, fissured, breaking into scales and often leading to a girdling of the twigs; subsequent development of cankers on branches and trunks circular, raised, persistent, and forming resinous scales thicker than bark, such lesions increasing in size and eventually girdling affected parts; leaf and fruit spots circular, dark brown, yellow-haloed, at times concentric, on fruit not penetrating beyond the albedo.

Differences do exist in the disease as it occurs in Argentina and in Florida, but these are minor, pertaining only to slight variations in the size, zonation, and number of spots on leaves, twigs, and fruit, and to the host range other than on citrus. These variations are regarded as of little significance and are considered to be effects of dissimilar environments or small differences in the causal organism.

We should like to take occasion here, in view of this reduction to synonymity of Florida scaly bark and lepra explosiva, to urge adoption of the name leprosis for both diseases as has been suggested by Fawcett. This becomes particularly desirable because the name scaly bark

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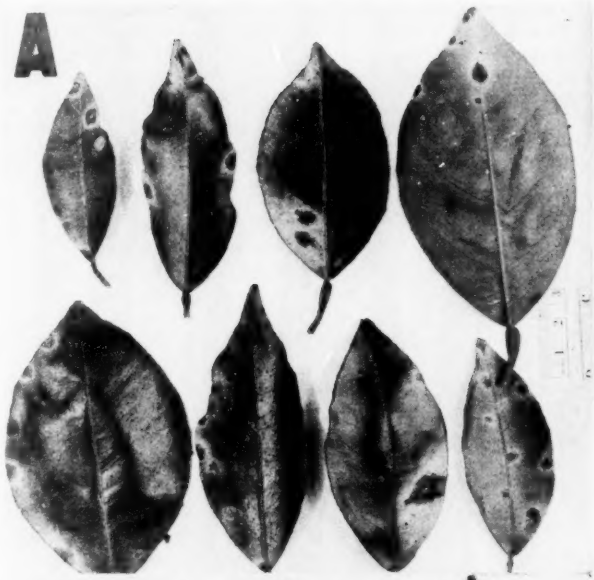
Table 1. Comparison between Florida Scaly Bark and Argentine Lepra Explosiva.

Characteristics	Florida Scaly Bark	Argentine Lepra Explosiva
I. Parts affected	I. Fruit, stems, leaves	I. Same*
A. On leaves		
1. Size	1. Spots small, averaging 1/4 inch.	1. Spots large, averaging 1/2 inch, one sometimes involving half of leaf.
2. Appearance	2. Spots circular, consisting of resinous brown platelets bordered by chlorotic halos.	2. Spots circular, resinous, the brown circles (sometimes numerous concentric ones) enclosing green tissue; chlorotic halo around outermost resinous circle.
3. Location	3. Spots predominantly (ca. 70%) marginal, not delimited by veins.	3. Spots not predominantly marginal, otherwise same.
4. Number of spots per leaf	4. Few to numerous (1 to 8).	4. Fewer on the average than Florida Scaly Bark (1 to 4).
5. Spots visible on:	5. Both sides of leaf.	5. Same.
6. First visible:	6. Summer and fall.	6. Same.
7. Leaf drop:	7. Yes.	7. Same.
B. On Stems	B. Original infections only on green twigs.	B. Same.
1. Size	1. Lesions pinpoint on immature shoots to girdling proportions on main branches and trunks.	1. Same.
2. Appearance		
a. Immature shoots	a. Lesions at first yellow pinpoint spots developing into larger (to 1/4 inch dia.) raised, reddish-brown spots.	a. Lesions at first yellow pinpoint spots developing into larger (to 1/8 inch dia.) raised, reddish-brown spots.
b. Mature shoots	b. Secondary development of spots, producing slight target-board effects; color yellow-brown to red-brown; at times coalescing to form scaly bark.	b. Secondary development of spots, producing distinct target-board effects; otherwise same.
c. Branches and trunks	c. Large circular raised persistent cankers with excrescent scales much thicker than healthy bark; undisturbed scales piling to form cone-like excrescenses. Size progressively increasing, eventually leading to girdling. Resinous streaks in wood under old lesions.	c. Same.
3. First visible:	3. Summer and fall.	3. Same.

Table 1. Continued

Characteristics	Florida Scaly Bark	Argentine Lepra Explosiva
C. On fruit		
1. Size of lesions.	1. Pinpoint to 1/4 inch dia. including halo.	1. Pinpoint to 3/4 inch dia.
2. Appearance		
a. Green fruit	a. Predominant form circular plates, at first yellow, later brown-centered with yellow halo; at times spots like predominant form under Argentine Lepra Explosiva.	a. Predominant form dark circular rings, slightly sunken, centers nearly normal, with halo around entire spots; at times spots like predominant form under Florida Scaly Bark.
b. Mature fruit	b. Spots circular, centers brown and sunken, occasionally fissured, often coalescing.	b. Same.
3. Location of spots	3. Generally distributed over fruits.	3. Same.
4. Number of spots per fruit.	4. 1 to 90.	4. 1 to 50.
5. Penetration of spots	5. Only into albedo.	5. Same.
6. First visible:	6. Summer.	6. Late summer.
7. Fruit drop:	7. Yes.	7. Same.
III. Hosts	III. Sweet orange-severely attacked. Sour orange-severely attacked. Grapefruit-very slightly attacked. Tangerine-very slightly attacked. (No other citrus varieties investigated.) Ligustrum (L. lucidum)-no symptoms.	III. Sweet orange-same. Sour orange-same. Grapefruit-not observed. Tangerine-very slightly attacked. (No other citrus varieties investigated.) Ligustrum (L. lucidum)-moderate infection. Ligustrina (L. sinense)-severe infection with marked leaf drop.
IV. Causal agent or vector		
A. Identity	A. Mite of the genus Brevipalpus.	A. Same (syn. Tenuipalpus).
B. Localization on citrus hosts	B. Mainly on striated twigs, only occasionally on leaves, fruit, and smooth green twigs.	B. Same.
V. Environmental relationships	V. Most prevalent along water courses.	V. Prevalent near water but frequent elsewhere.
VI. Control	VI. Sulfur spray program	VI. Same.

* The word "same" refers to a similarity with Florida scaly bark.



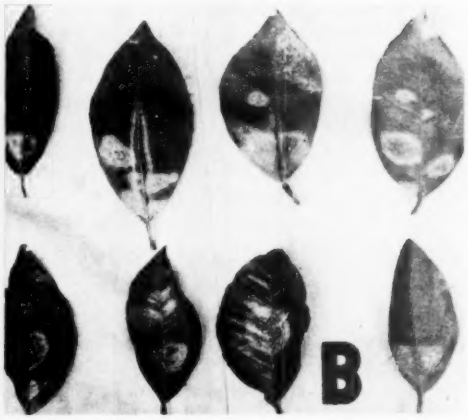
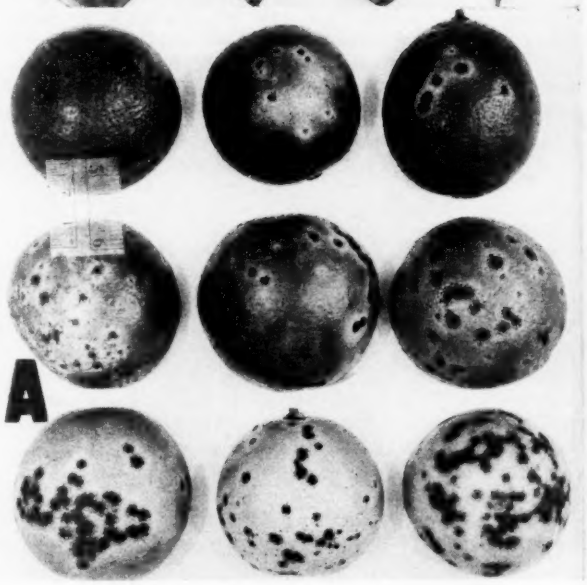


FIGURE 1. Leaf symptoms.
A. Florida Scaly Bark
B. Argentine Lepra Explosiva



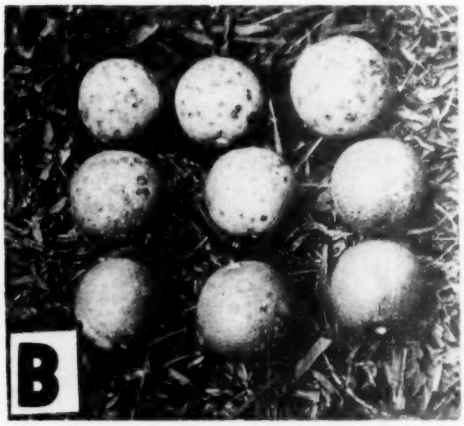


FIGURE 2. Fruit symptoms.
A. Florida Scaly Bark
B. Argentine Lepra Explosiva

refers in Florida to the disease under discussion, and in California to an entirely different trouble, namely psorosis. The term leprosis is further to be preferred since it is more descriptive of lesions on both fruit and bark than either of the two Florida names scaly bark and nailhead rust, whose connotations refer to one or the other but not to both aspects of the disease. Furthermore, the word leprosis already has some acceptance in Argentina and is not likely to be confused with any other name in the field of plant pathology.

Since it is evident that both diseases are synonymous, the question now arises of what significance is this relationship to the Florida grower? Does the presence of an identical disease so destructive in South America indicate that there is any imminent danger here?

To answer this question, it will be interesting to review briefly the history of leprosis in Florida. It was in Pinellas County in the 1860's that the disease was first observed. Here, in

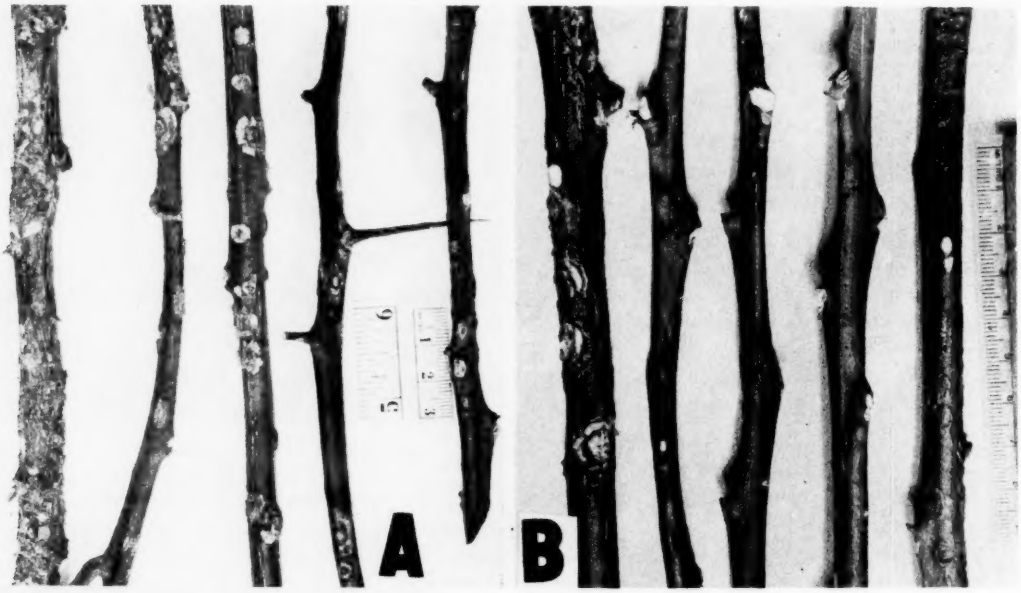


FIGURE 3. Twig symptoms.
A. Florida Scaly Bark B. Argentine Lepra Explosiva

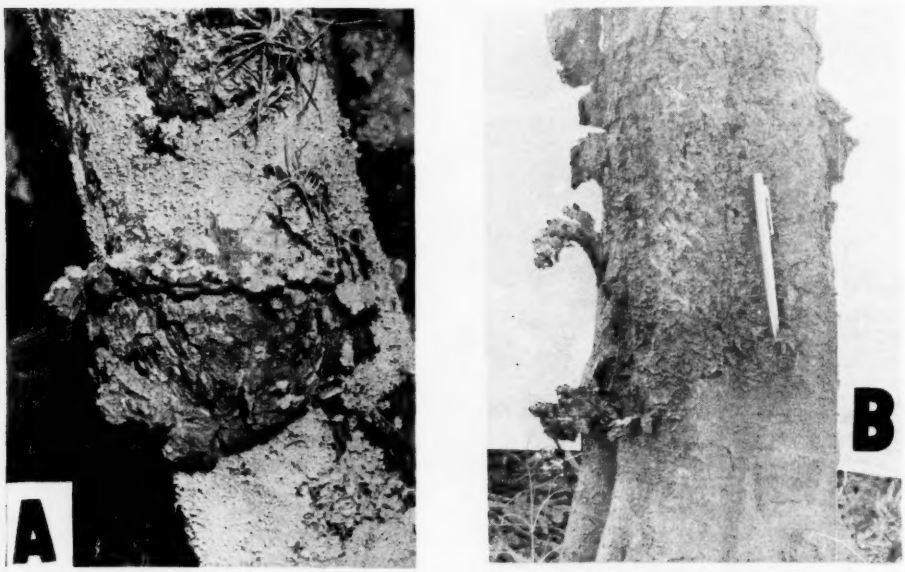


FIGURE 4. Trunk symptoms.
A. Florida Scaly Bark B. Argentine Lepra Explosiva

the old Phillippi grove on the shore of Safety Harbor, leprosis appeared in one of the original seedling trees. The trouble spread slowly over several acres of grove and then jumped into an adjoining nursery. In later years many of the groves in the surrounding country planted to stock from this nursery were reported to have developed severe cases of leprosis. Dissemina-

tion continued until by 1906 it had become so widespread and of such importance that a project for its control was initiated by the Florida Agricultural Experiment Station. No abatement in importance occurred, and by the time of the years of World War I, leprosis had already moved across the Florida Peninsula and was to be found in nine counties, ranging from Pinellas on the West Coast to Brevard and Volusia on the Indian River. Apprehension concerning its destructiveness ran so high that the State Plant Board finally took vigorous steps to curb further spread by promulgating the Public Notice of March 12, 1917, in which was listed 120 towns in nine counties as being covered by Rule 15, which read in part:

> "The movement of citrus trees, or parts thereof, from the areas designated in the Public Notices by the State Plant Board as areas in which citrus trees are likely to be infected with scaly bark, into all parts of the State of Florida other than those specified as infected areas, is hereby prohibited."

By 1925 the trouble had nevertheless spread to 17 counties including Pinellas, Pasco, Hernando, Hillsborough, Sumter, Lake, Polk, DeSoto, Okeechobee, Osceola, Orange, Seminole, Alachua, Putnam, Volusia, Brevard, and St. Lucie, but by then leprosis seemed to have passed its climax, and in that year the State Plant Board, in view of "careful studies which have been made of the scaly bark situation" and because of "more than 10 years of observation and experience with the disease", revised its Rule 15 downward to read, in part: "the Nursery Inspector shall not in any case issue certificates of inspection ..... to cover the citrus stock in any nursery adjacent to a known scaly bark infection." This ruling, unchanged since its promulgation, is the one in force to the present time.

The status of leprosis in Florida today is one rather unique among plant diseases. It is the story of a malady that with the passing of the years has virtually disappeared. Today there is known to exist in the State but one small pocket of leprosis, and that in a few unsprayed jungle groves on the East Coast.

The question as to why leprosis has become practically extinct cannot be answered save by inferences. Factors contributing to its disappearance are not known with certainty but several influences can be suspected such as climatic changes and changes in cultural practices, both as they affect a causal agent.

With regard to the nature of the causal agent, work in Argentina and at the Florida Citrus Experiment Station indicates that the disease is associated in some way with a mite of the genus Brevipalpus. It is not yet clear whether the mite itself causes the lesions by the injection of a toxin or whether the mite functions as the vector of a virus, but this much is certain: that mites induce it, and that control is obtained wherever sulfur sprays or dusts are employed. This is true for the condition both as it exists in Argentina and in Florida.

While climatic changes with their attendant influences on the causal agent, its cycles, and its biotic environment, have certainly had some bearing on the question, it appears more likely that the decline in leprosis was brought about by modifications within recent decades in the care of the Florida groves, particularly in the matter of spraying and dusting. The spray crew now visits a grove with the same regularity as the fruit picker, whereas 35 years ago, at the time when scaly bark was rampant, spraying was considered more or less a rich man's fancy. Spraying and dusting first began to take the proportions they assume today during the latter years of this century's second decade. This, as it happens, coincides quite closely with the time leprosis began to level off and disappear. The correlation is further strengthened by virtue of the leprosis mite's marked susceptibility to the very sulfur toxicants so extensively used since that time.

While there may be other explanations for the disappearance of leprosis, there is one point that is fixed and certain: If the leprosis mite does return, the use of sulfur will control it. By the same token however, regarded reversely, if the now widespread practice of spraying should ever materially decrease, if large acreages go long unprotected, as is advocated by some who would abandon altogether the spraying of canning plant fruit, then there is certainly a fair degree of probability that leprosis mites together with other pests waiting at the fence row, will be ready to invade such unprotected groves, increase their kind, spread, and once again become the serious problem of yesteryear. The next era of low citrus prices with its undoubted reduction in pest control practices, coupled with the canning-plant philosophy of fruit growing, may well see re-entry of such an old-time trouble as now visits the citrus industries of Paraguay and Argentina.

TRISTEZA LABORATORY (Florida State Plant Board) ESTACION EXPERIMENTAL CITRICOLA CONCORDIA, ENTRE RIOS, ARGENTINA

PHYGON XL FOR THE CONTROL OF PEACH BLOSSOM BLIGHT

Dwight Powell

Studies for the past several years have emphasized the importance of blossom blight control on peaches in reducing brown rot (Sclerotinia (Monilinia) fructicola) incidence at harvest. Due to the erratic nature of blossom blight infections it has been difficult to secure significant data in Illinois. In 1949 and 1950 different orchards were given identical treatments with the hope of securing at least one orchard which would have sufficient infection for a comparative test. It is felt that the following data de-emphasize the importance of the pink bud application, emphasize the efficacy of Phygon XL in preventing blossom blight infections, and stress the importance of blossom blight control in reducing preharvest incidence.

Methods: In 1949, single tree plots were arranged at random on Red Bird with eight replications per preatment. On April 1, in the pink bud stage, the grower applied liquid lime sulfur, 1 gallon plus sulfur paste, 4 pounds to 100 gallons of water. On April 5, in the early bloom period, the treatments listed in Table 1 were applied. On April 29, 100 blossoms were selected at random from each tree and the number of infected blossoms recorded. Only those blossoms showing conidia were considered infected. Laboratory isolations have shown, however, that 10 to 25 percent of the so-called weak blossoms (those which fall normally and do not set fruit) are infected but do not develop conidia.

Table 1. Brown rot blossom blight infection, 1949.

Treatment to 100 gallons of water		Mean blossom infection
Pink, April 1	Bloom, April 5	
Liquid lime sulfur, 1 gallon Sulfur paste, 4 lbs.	Phygon XL, 1/2 lb.	5.8
"	Ferbam, 1 lb.	13.7
"	Bioquin 1, 1/4 lb. Sulfur, 3 lbs.	14.9
"	CR 305, 1 lb.	15.4
"	Ziram, 1 lb.	15.5
"	Sulfur paste, 8 lbs.	18.0
"	None	28.0
L.S.D. at 5% level		7.8

Table 2. Brown rot infection, 1950.

Treatment to 100 gallons of water	Mean blossom infection May 12	No. blight cankers per 100 twigs May 12	Percent fruit infection 7-24	8-8	8-14
Phygon XL, 1/4 lb. Wettable sulfur, 3 1/4 lbs.	1.0	2.6	1.45	7.0	6.40
Wettable sulfur, 8 lbs.	7.0	5.0	2.00	11.0	17.00
Check, no treatment	15.1	7.1	--	--	--
L.S.D. at 5% level	1.4	1.7	.18	.98	9.6

In 1950, a fifteen-acre block of Elberta was divided into three equal plots and on April 18, in the early to full bloom period, was given the treatments listed in Table 2. The check plots received no treatment during the entire season and consequently had lost all of its fruit by July 24. The other two plots received the same treatment for insect control after the blossom application. On May 12, the mean blossom infection was recorded by examining 100 blossoms from each of 8 trees per plot. Only those blossoms showing conidia were considered infected. Also, on May 12, the number of active blight cankers to 100 twigs of each of the same 8 trees per treatment were recorded. On July 24, August 8, and August 14, the same trees were examined for fruit infections by observing 100 fruits per tree. All data were analyzed by an analysis of variance.

Results and Discussion: In Table 1, it is shown that the pink bud spray was not too effective in reducing blossom blight although it is possible that the check plot would have had more than 28 percent infection had this spray been omitted. Phygon XL was particularly effective in allowing only 5.8 percent infection and was significantly better than the other treatments. All of the treatments, however, reduced blossom infection below the check. None of the treatments caused injury, although Phygon XL seemed to have discolored a number of the blossoms. For this reason, it was reduced to 1/4 pound in the 1950 test, and sulfur, 3 1/4 pounds, was added to give the spray a little more potency.

In Table 2, it is shown that the treatment containing Phygon XL and sulfur was superior to sulfur alone both in the number of blossoms infected and in the number of active twig cankers to develop conidia. Inasmuch as twig cankers are the primary source of inoculum after the bloom period, a small number of such cankers can develop a serious brown rot epidemic providing, of course, weather conditions are conducive to infection. In Table 2, it is shown that the canker ratio May 12 between the two treatments was approximately 1 to 2. At harvest, August 14, the fruit infection ratio between the same two plots was approximately 1 to 3. Thus, canker abundance early in the season has a definite bearing on the severity of fruit infection at harvest.

Summary: In 1949 and 1950, sprays containing Phygon XL were superior to other treatments in reducing blossom blight infection on peaches. By increasing the control of such infection during the blossom period it was possible to retard the rate of development of the preharvest fruit infection.

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WITCHES' BROOM OF ALFALFA IN THE SOUTHWEST

Bailey Sleeth

Witches' broom of alfalfa was first reported[1] in 1944 in the Southwest from a single farm on the Yuma Mesa five miles south of Yuma, Arizona. It has been reported[2] as occurring in Washington, Oregon, Idaho, Utah, British Columbia, Alberta and Australia. It is interesting to note that areas from which witches' broom have been reported are widely separated. This suggests the possibility that more than one virus or virus strains are involved or that the disease has been transmitted from its point of origin to the other areas in some unusual manner.

Witches' broom has spread from the small localized area from which it was reported on the Yuma Mesa in 1944 to farming areas up to 30 miles in distance in opposite directions, -- to the east and west. These two areas are separated from the point of initial infection by at least 25 miles of desert land. The disease has greatly increased on the Yuma Mesa and has become an important factor in reducing yield of both hay and seed. Infected plants soon become stunted, fail to produce seed and thin out the stand by dying. In a survey made in October 1950, alfalfa plants with typical witches' broom symptoms were found throughout the 5000 acres of alfalfa on the Yuma Mesa, in the Yuma and Gila Valley areas adjacent to the Yuma Mesa, and in the Wellton-Mohawk Valley at a point 30 miles east of Yuma. In California witches' broom was found in the Bard Valley, which lies across the Colorado River from the Gila Valley, and on a Bureau of

[1] Hoyman, W. G. Alfalfa diseases in Arizona and New Mexico. Plant Dis. Reptr. 28: 291-292. 1944.

[2] Menzies, J. D. Witches' broom of alfalfa in North America. Phytopath. 36: 762-774. 1946.

Reclamation pre-development farm located on the East Mesa, 30 miles west of Yuma. The heaviest infection was found on the Yuma Mesa, where in some instances 25 percent of a stand was estimated to be infected. Infection in the other areas was much lighter, -- less than 1 percent.

The increase in amount and spread of witches' broom has been rapid on the Yuma Mesa since it was first reported six years ago. It is estimated that infections in stands comprising the 5000 acres of alfalfa range from around 2 percent to around 25 percent of the stand and are increasing. The percentage of infection is greatly influenced by age and density of stand. Infection is highest in the older and less dense stands. In such stands the thinned-out condition is in part due to witches' broom infection. A sample plot count was made in three representative fields of Hairy Peruvian alfalfa. Infected plants in 20 plots, 0.001 acre in size, were counted in each field. In two fields with five-year-old plants an average of 9,550 and 10,200 infected plants per acre were found. These stands were thin and approximately 15 to 20 percent of the plants were infected. In the third field of four-year-old alfalfa 3,800 infected plants per acre were found. This represents an infection of 2 to 5 percent as the stand was fairly dense.

About-midsummer of 1950 witches' broom was observed in an alfalfa variety test which had been planted with Hairy Peruvian, India, African and Chilean in March 1949. The planting consisted of two stocks of four varieties each planted in double rows, 100 feet long and replicated four times. The two stocks of each variety represented a different seed source. This planting afforded an opportunity to compare the apparent susceptibility of the four varieties. The results of a count made in October 1950 are given in Table 1.

Table 1. Witches' broom infection of four varieties of young alfalfa.

Variety		Plants with witches' broom	
		Per Plot	Per Acre
		Number	Number
Hairy Peruvian	1	16.5	2871
Hairy Peruvian	2	17.0	2958
India	1	14.2	2470
India	2	13.5	2349
African	1	16.0	2784
African	2	10.2	1775
Chilean	1	13.0	2262
Chilean	2	13.0	2262
L.S.D. at 5% level		5.1	

A casual glance at the results given in Table 1 would seemingly indicate that there is a difference in susceptibility between one or more varieties, or seed stocks. It is hoped that this is the case, but these data may be misleading in that symptoms of witches' broom may be more readily recognized in one variety than in another. This difficulty may be more pronounced in a young stand than in an old alfalfa stand. Also, survival of infected plants would be a better measure of resistance than number of plants with symptoms. Perhaps the most important aspect of these results, Table 1, is that the incidence of witches' broom is so high in a stand less than two years old. This is believed to be the result of one or two conditions or both: (1) that there has been an enormous increase of the disease or inoculum in the area and (2) that vectors are plentiful. The first factor is self-evident in that the disease has increased and the second factor is highly probable in that potential insect vectors are abundant. While no insect vectors have been identified in this area, the possibility of one or more is good. The insect population of potential vectors, as aphids, leafhoppers, stink bugs, lygus and other insects is high and causes heavy losses especially to alfalfa seed producers.

Discussion and Summary: -- At present witches' broom is one of the most important alfalfa diseases on the Yuma Mesa where 90 percent of the land is in alfalfa production. If the present trend of increase continues it will soon become the most important disease in this area. The present agronomic practice is to hold stands as long as they are productive and renovate and reseed rather than destroy the old stand by plowing and reseeding. This permits a heavy build-up of the disease and a continuous reservoir of inoculum. If it becomes necessary as a control measure to plow and reseed every two or three years, this added expense will seriously jeopardize the economic existence of many of the homesteaders on the Yuma Mesa. It would be well not to overlook the possibility of developing a resistant selection or variety if long-lived productive

stands are desired.

DIVISION OF SOIL MANAGEMENT AND IRRIGATION, BUREAU OF PLANT INDUSTRY, SOILS AND AGRICULTURAL ENGINEERING, U. S. DEPARTMENT OF AGRICULTURE

CURVULARIA LEAF BLIGHT OF LADINO CLOVER

S. G. Lehman

In October 1948, leaf infections not hitherto observed by the writer were seen in Ladino clover plots growing at Plymouth, North Carolina. Fully 5 percent of the leaves bore infections on one, two, and sometimes all three leaflets of each leaf. Microscopic examination of the diseased tissue revealed the presence of fungus hyphae growing scantily on and in the diseased tissue. After a few days in a moist culture dish numerous short brown conidiophores with conidia of the genus Curvularia were found.

Isolations were made by using single Curvularia-type conidia washed from old lesions, and by plating surface-sterilized tissue from young lesions. The fungus most frequently obtained from the young lesions was the same as that obtained by isolating single conidia of the Curvularia type obtained from the surface of older lesions. A Colletotrichum with straight spores borne in setose acervuli was also obtained from some of the lesions which yielded the Curvularia fungus.

Inoculations with the two types of fungi isolated were made on Ladino clover plants grown from seed in pots in the greenhouse. Cultures of each fungus growing on potato dextrose agar were ground to a thin paste in a mortar and used for inoculum on leaves and stems of the clover plants. Uninoculated plants were left for controls. Moisture conditions were maintained at a high level for 60 hours following inoculation, then at the usual greenhouse humidity level with the temperature varying from a mean of 70° F. at night to 85° F. by day.

No infections occurred at any time on the uninoculated control plants nor on those inoculated with the straight-spored Colletotrichum. Nine days after inoculation with each of two isolates of Curvularia, definite symptoms of infection were present as small chlorotic areas and larger watery brown areas on the leaflets and petioles of inoculated plants. The Curvularia fungus used for inoculation was recovered by reisolations made from the artificially infected leaflets and petioles.

In 1920, Bonar[1] described a fungus wilt of white clover and the name Brachysporium trifolii Kauff. was given to the pathogen. The fungus obtained from diseased Ladino clover leaves in North Carolina and subsequently shown to be pathogenic on that suscept agrees in general cultural characters and conidial size, shape, septation, and color with those of Brachysporium trifolii as given by Bonar. This fungus has since been transferred to the genus Curvularia and is now known as Curvularia trifolii.

In 1924 Bonar[2] published results of further studies with this fungus. In greenhouse inoculation tests he established the pathogenicity of the fungus on several species of Trifolium including T. repens lotus (Ladino variety). He noted that 75 percent of the leaves became infected and that infection progressed more rapidly on this than on the other varieties tested.

FIGURE 1. Curvularia leaf blight of Ladino Clover.

Lesions produced by Curvularia trifolii on Ladino clover may occur on leaflets or petioles. Infections commonly occur at the tips or margins of leaflets and advance inward. Infected tissue is chlorotic at first but soon becomes light brown, watery, more or less translucent with chlorosis at the advancing edge. Diseased areas arising at leaf tips are commonly V-shaped (Figure 1). Under conditions of high moisture, the fungus rapidly invades the entire leaflet and grows down the petiole. When petiole infection occurs, the leaf takes on a wilted appearance, collapses and is soon hidden from view by healthy leaves. The true extent of damage is often not apparent until the observer pushes aside the top layer of more or less healthy foliage and notes the many brown dead prostrate

[1] Bonar, Lee. Wilt of white clover due to Brachysporium trifolii. Phytopath. 10: 435-441. 1920.
[2] _____. Studies on the biology of Brachysporium trifolii. Amer. Jour. Bot. 11: 123-156. 1924.

leaves below. Large numbers of conidia may be found on these shaded dead leaves. The conidia are brown with lighter colored, basal and terminal cells, mostly 3-septate, usually slightly or markedly bent at the penultimate cell and bear a very short stipe.

This disease has been observed in Ladino clover in North Carolina at Raleigh and Plymouth each year since 1948. It develops rapidly during wet warm periods in fall, winter and spring. It seemingly does little damage in fields that are closely pastured or frequently mowed, but in areas along roadways or fences where the foliage is allowed to stand for long periods 20 to 25 percent of the leaves have been observed to be diseased and a high proportion of them dying. Apparently a good degree of control may be obtained by pasturing or mowing to prevent excessive shading due to accumulation of foliage.

UNIVERSITY OF NORTH CAROLINA, RALEIGH

ANTHRACNOSE RESISTANCE IN LUPINES[1]

J. L. Weimer

Anthracnose (Glomerella cingulata) is one of the most important diseases of the blue lupine (Lupinus angustifolius L.) in certain areas of the lupine belt in the southeastern United States. The fact that the usual methods of plant disease control have proved to be inadequate has stimulated the search for resistant varieties of lupine both in locally grown strains and in varieties and species from abroad. Until recently all attempts by the writer to find resistance gave negative results. That resistance to anthracnose does exist, however, is shown by the results of inoculation experiments conducted during the past few months. Up to the present time resistance has been demonstrated only in plants of some imported strains of blue lupine and in one strain of yellow lupine (L. luteus L.). Resistance in these plants has been demonstrated only in the early vegetative stage. No determination has been made as to whether resistance is retained as the plants approach maturity, or whether the pods and seeds are resistant.

Four experiments have been conducted in which 47 seed lots were tested one or more times. Six locally grown selections and 23 imported strains of blue lupine, 10 lots of yellow lupine, and 8 of white lupine (L. albus L.) were included in these tests. The plants were grown in pots in the greenhouse or in short rows out of doors. Ten to 50 seeds of each lot were planted, the number depending on the quantity of seed available.

Several methods of inoculation were used. These included spraying the plants with a spore suspension or a suspension of macerated mycelium, inoculating the seeds before planting by dipping them into a heavy spore suspension, and inserting a droplet of spore suspension into the cotyledons or stems with a hypodermic needle. The incidence of infection varied with the moisture conditions even when the inoculum was inserted directly into the tissue. Usually 100 percent infection was obtained when the seeds of susceptible plants were inoculated if they were planted in moist soil and there was sufficient moisture to permit infection, sporulation, and the spread of the fungus. No infection was obtained by this or any other method during dry weather.

Although the data from these experiments will be published elsewhere, a general summary of the results will be given here. All plants of the commercial blue lupine and six selections from it were extremely susceptible. Under conditions which brought about rapid death of these plants only minute lesions developed on the stems, cotyledons, and leaflets of plants of some of the imported lots of blue lupine and one of yellow lupine. Lesions sufficiently large to girdle and kill a plant occasionally developed in some lots; but usually only a few of the lower leaflets were shed, and the disease disappeared with the return of dry weather. Eight of the most resistant lots of blue lupine and one of yellow lupine were selected, replanted, and retested. Since the hypodermic needle method of inoculation gave the most consistent and uniform infection, this method was used in the final test. One hundred percent of the plants of the commercial blue lupine used as susceptible controls developed large cankers and most of them were girdled. The stems of a few plants of some of the imported lots developed small cankers but only a small number were girdled. For the most part plants of the imported lots developed no lesions at the point of inoculation.

The results of these experiments indicate that under the existing conditions the following imported lots, although not immune, were highly resistant to anthracnose at least in the early

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[1]Paper No. 204, Journal Series, Georgia Experiment Station.

stage of growth: blue lupine -- P.I. 167938, P.I. 167943, P.I. 168529, P.I. 168534, and P.I. 168535; and yellow lupine -- P.I. 168539. All of these introductions are from Portugal.

These imported strains of blue lupine make a fair growth but are not so vigorous as is the strain now grown commercially. They blossom about two weeks later and have somewhat smaller seeds than does the local strain. The seeds are moderately to very hard and must be scarified before being planted. The lack of vigor and the hard seeds probably will make these varieties unacceptable to the farmer. They are, therefore, considered at present only as a good source of anthracnose resistance. These importad lots are not highly resistant to brown spot (Ceratophorum setosum) but are more resistant than some varieties and species now grown commercially. So far as known at present these imported blue lupine varieties are as winter hardy as the strains now grown in the South.

There has been no opportunity as yet to increase the seed of these resistant lots. However, the writer has a small quantity of seed of each lot and will send a few seeds to any lupine breeder who may desire them.

COOPERATIVE INVESTIGATIONS OF THE DIVISION OF FORAGE CROPS AND DISEASES, BUREAU OF PLANT INDUSTRY, SOILS, AND AGRICULTURAL ENGINEERING, AGRICULTURAL RESEARCH ADMINISTRATION, U. S. DEPARTMENT OF AGRICULTURE, AND THE GEORGIA EXPERIMENT STATION

ASCOCHYTA CANKER OF BLUE LUPINE[1]

J. L. Weimer

During late winter and spring of 1949 and 1950 numerous blue lupine (Lupinus angustifolius L.) plants growing in experimental plots at Experiment, Georgia, were killed by a disease. The same disease was found in lupine plants growing in commercial fields in several locations in central Georgia. This disease always occurred in lupines that had been planted on land on which cotton had grown the previous summer. The percentage of diseased plants usually was small but in limited areas it was sometimes high enough to reduce the stand seriously. Plants of all ages appeared to be susceptible.

Careful examination of the diseased plants showed dark brown cankers, one to three inches long, near the bases of their stems. At first these cankers were assumed to be caused by the anthracnose fungus (Glomerella cingulata). More careful examination, however, revealed the presence of pycnidia more or less abundant over the surface of the cankers. The fungus was isolated and studied. Pycnospores from the host were hyaline, 0-1 septate, straight or slightly curved, rounded at the ends, and typically not constricted at the septum. Fifty 1-septate spores from the host measured 3.2 - 4.2 x 8.4 - 10.5 microns (Av. 3.9 x 9.2 microns).

The close correlation between the presence of the disease and of old cotton stalks suggested that both the lupine and cotton might be hosts. A study of the fungus showed that the spores were very similar to if not identical with those of Ascochyta gossypii[2].

Inoculations were made on cotton and lupine plants growing in the greenhouse by atomizing the plants with blended mycelium from a pure culture of the fungus isolated from lupine and holding them in a moist chamber for 48 hours. Five days after inoculation the cotton leaves had numerous circular lesions about one centimeter in diameter. The lesions had a greenish yellow to grayish center and a light to dark brown border. Petioles and squares also became infected, but no lesions were seen on the stems. In the lupine, infection appeared only on the cotyledons. These lesions were circular, brownish in color, and in some instances involved a large proportion of the cotyledon. Judging from this single experiment, this fungus is a vigorous parasite on cotton but a weak one on lupine. Just how infection in the lupine takes place is not known. The fungus may act largely as a wound parasite or it may enter the stem from infected cotyledons, but further observations and study are necessary to determine this point.

Since this disease appears to be limited largely to lupines in fields in which cotton has been growing and since the causal fungus appears to be identical with that causing Ascochyta blight of cotton, the fungus causing the disease on these plants may be considered to be identical. To what extent, if at all, growing lupines in fields to be planted to cotton will increase the incidence

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[1]Paper No. 205, Journal Series, Georgia Experiment Station.

[2]The writer wishes to express his appreciation to Dr. G. E. Thompson, University of Georgia, Athens, Georgia, who studied the cultures used in these investigations and confirmed the tentative identification made by the writer.

of Ascochyta blight in cotton remains to be determined. At least in some areas corn and peanuts rather than cotton are planted after lupines. This crop sequence should serve at least as a partial control measure in so far as the Ascochyta disease of cotton is concerned. On the other hand it is a general practice to plant lupine in the cotton fields, often without much preparation. Smith[3] has recently pointed out that the severity of Ascochyta on cotton appears to be proportional to the old plant material from the previous cotton crop remaining on the surface of the soil. Assuming that the disease was present and that the old cotton stalks are simply disked down before the lupine seed is sown, there should be ample inoculum present to initiate the disease in the lupine when the weather conditions are suitable for infection. Turning under the cotton stalks completely should, according to Smith's observations on cotton, give at least partial control of the disease on lupine.

COOPERATIVE INVESTIGATIONS OF THE DIVISION OF FORAGE CROPS AND DISEASES, BUREAU OF PLANT INDUSTRY, SOILS, AND AGRICULTURAL ENGINEERING, AGRICULTURAL RESEARCH ADMINISTRATION, U. S. DEPARTMENT OF AGRICULTURE, AND THE GEORGIA EXPERIMENT STATION

SEED TREATMENT AND SEED INOCULATION STUDIES WITH SOYBEANS

T. J. Nugent, S. B. Fenne, and W. C. White

In order to check the effect of combining seed inoculation and seed treatments to soybeans, a field test was made at Norfolk, Virginia in 1950. A bulk lot of Ogden soybean seed was divided into four parts, two parts of which were treated with Arasan (3 ounces per 100 lbs.), and two parts of which were left untreated. On May 9 one lot of each of the treated and untreated seed was inoculated with Nitragin and all treatments planted immediately afterwards. The soybeans were planted 3 inches apart in rows 3 1/2 feet apart. Each treatment was replicated (100 seed per replicate) four times in a Latin square arrangement. This Latin square was duplicated and the data, as given in the following tables, are averages of the two Latin squares (eight replications).

Stand counts were made on three separate dates (May 23 and 31, and June 14) and the average germination for each treatment on the three dates is shown in Table 1. When the data were analyzed statistically it was found that the differences between the treatments were highly significant. The Arasan treatment, when used alone, gave stand counts significantly higher than for the treatments in which Nitragin was used alone, and for the control. The Arasan plus Nitragin treatment was a close second to Arasan alone, except for the slight delay in emergence as shown by the count on May 23 (Table 1).

Table 1. Average germination for each treatment by dates.

Treatment	May 23	May 31	June 14
Nitragin	60.4	66.8	66.8
Arasan	71.6	77.9	77.8
Arasan + Nitragin	62.6	76.1	75.9
Control	49.1	61.1	60.5
L.S.D. at 5% level	9.6	7.4	8.0
L.S.D. at 1% level	13.0	10.1	10.9

On November 2 the soybeans were harvested with a small soybean thresher. Using 60 pounds as the weight per bushel, the average calculated yields in bushels per acre are given in Table 2. The yield differences between treatments were not significant.

As far as could be determined, the field in which the soybeans were grown had not been planted to soybeans previously. Examination of the roots of the beans at harvest time did show some root nodulation on all replicates regardless of treatment. The plants from the Nitragin-treated seed, however, had considerably more nodulation than did those from seed not receiving

[3]Smith, A. L. Ascochyta seedling blight of cotton in Alabama in 1950. Plant Dis. Reptr. 34: 233-235. 1950.

Table 2. Average yields in bushels per acre by treatments.

Treatment	Bushels per acre
Nitragin	40.13
Arasan	41.05
Arasan plus Nitragin	42.02
Control	40.92
L.S.D. at 5% level	N.S.

the Nitragin treatment.

Conclusions: The use of the Arasan seed treatment significantly increased the stand counts of the soybeans in this test. Rood nodulation was heavier on the plants grown from seed which had been inoculated with Nitragin just prior to planting. Although Arasan plus Nitragin increased the average acre yield approximately one bushel over the control and about two bushels over Nitragin alone, the yield differences between treatments were not significant.

EXTENSION SERVICE, VIRGINIA POLYTECHNIC INSTITUTE, BLACKSBURG

DISEASES OF TALL FESCUE GRASS IN GEORGIA[1]

E. S. Luttrell

During recent years tall fescue grass (Festuca elatior var. arundinacea (Schreb.) Wimm.) has been planted extensively in Georgia and has become the most important permanent pasture grass for winter grazing in the Piedmont and in North Georgia. It is also utilized, at least in North Georgia, for grazing and hay during the summer. Disease surveys of forage crops in 1949-50 indicate that fescue is subject to attack by several diseases, some of which are serious enough to make the development of control measures desirable. Fescue apparently has a high nitrogen requirement, and lack of an adequate supply of nitrogen is one of the factors most commonly responsible for decreased production of fescue. Netblotch is the most important disease during the winter and early spring when the grass is being used for grazing. During the summer, Rhizoctonia leaf blotch is by far the most serious disease. Other leaf diseases are Cercospora leaf spot, which appears to be potentially serious, and spot blotch, which is uncommon and of minor importance. Rust has not been found on tall fescue, although it occurs on meadow fescue (Festuca elatior L.) and F. gigantea (L.) Vill. In addition to these leaf diseases, crown rot and white mold have been observed on fescue. Although the latter diseases may result in the death of entire plants and consequently might be expected to be of greater importance than the leaf diseases, they have been observed to cause only occasional damage. Drought and overgrazing appear to be of greater importance in the reduction of stands. No diseases directly affecting the inflorescence have been observed. Although the majority of these diseases occur during the summer, they must be given consideration, even in areas in which the grass is used only for winter grazing, since they may affect plant vigor and survival as well as the accumulation of forage for early fall grazing.

Nitrogen Deficiency. One of the most frequently encountered diseases of fescue was characterized by a yellowing of the leaves accompanied by the gradual decline of the entire plant. The older leaves first exhibited symptoms, becoming progressively yellowed and dying back from the tips. The premature death of the older leaves and the faded color and reduced growth of new leaves resulted in a high proportion of dead tissue and a poor appearance of the entire stand. In fertilization tests with fescue, conducted by Dr. O. E. Sell and E. M. Parker, this condition was pronounced in plots that received no nitrogen, whereas it was largely corrected in plots that received heavy applications of nitrate. The diagnosis of this disease as nitrogen deficiency was confirmed by leaf tissue analyses made by Dr. L. C. Olson. Since the leaf spots caused by fungi also result in the yellowing and dying back of older leaves, it is possible to overestimate

[1]Paper No. 207, Journal Series, Georgia Experiment Station.

the damage caused by these diseases in poorly fertilized stands unless the effects of nitrogen deficiency are taken into consideration. Even so, it is often difficult to distinguish between the proportion of damage caused by leaf spots and that resulting from nitrogen deficiency.

Netblotch (Helminthosporium dictyoides Drechs.). Infection with netblotch occurred throughout the State and reached its peak in the late winter and early spring of 1949-50. Although as many as 90 percent of the leaves were infected in some plantings, estimates of damage varied only from a trace to 5 percent. The disease was not present during the summer of 1949, the first evidence of infection being found about the first of December. However, it was present in trace amounts in all parts of Georgia throughout the summer of 1950.

The most important diagnostic feature of the disease was the appearance in the leaf tissue of short, irregular, dark-brown transverse bars which resembled short strands of dark thread drawn across the leaf. These bars, combined with short longitudinal streaks of brown tissue, produced an obscure network. The bars and streaks were dispersed, covering large, indefinitely limited areas of the leaf with an indistinct net, or aggregated to form brown blotches. Frequently the lesions were distinctly limited, rectangular, dark-brown, solid spots measuring 5-28 x 1-1.5 mm., and only the barest traces of a network were discernable at the ends of the spots. Infected leaves finally yellowed and died back from the tips.

Conidia of the causal fungus were formed over the dead leaf tips and developed abundantly when the leaves were kept in a moist chamber for 24 hours. In its morphology the fungus corresponded with the description given by Drechsler (Jour. Agr. Res. 34: 641-740. 1923) for Helminthosporium dictyoides. Conidia from Georgia material measured 56-115 x 14-19.6 microns (Av. 25 = 84.9 x 16.5 microns). H. dictyoides was isolated from conidia and from infected leaf tissue. No other fungus was obtained from tissue plantings made during the winter. However, during the summer, Colletotrichum graminicola (Ces.) G. W. Wills. and a species of Alternaria belonging to the A. tenuis group were the organisms most frequently isolated. In culture on 2 percent malt agar H. dictyoides produced a fluffy, pale gray mycelium which ultimately became almost black at the surface of the agar and formed small, spherical, black sclerotia. A fringe of tiny erect clavae composed of compacted hyphae developed around the margin of the colony where it was in contact with the walls of the culture tube.

Rhizoctonia Leaf Blotch (Rhizoctonia solani Kühn). Rhizoctonia leaf blotch was a serious disease on fescue throughout Georgia during the summer of 1949. Estimates of the reduction in leaf area it caused at various locations during July and August varied from 10 to 40 percent. The peak of infection was past by September and by November only traces of the disease remained. During the drier summer of 1950, it was much less abundant. Because of a drought during June, there was hardly any evidence of the disease until July, and then infections were scattered. The highest estimate of damage caused was 5 percent.

The lesions were large, irregular, grayish-tan blotches outlined by fine, dark-brown borders. Under the hand lens the appressed, pale tan mycelium of the causal fungus was visible over the surface of the lesions, and in humid weather the mycelium developed into large, white, cottony tufts of hyphae. The blotches often coalesced to cover large areas of the leaf blade. Similar blotches occurred on the leaf sheaths. The leaves finally died and turned brown. The perfect stage of the fungus was found at several locations. It appeared as extensive, thin, mealy, white patches on the lower surface of green healthy leaves and in structure agreed with the description of Pellicularia filamentosa (Pat.) Rogers published by Rogers (Farlowia 1: 95-118. 1943). Basidiospores of the fungus on fescue measured 7.3-9.8 x 4.6-6.2 microns (Av. 25 = 8.3 x 5.6 microns). Isolates of the fungus were obtained from infected leaf tissue and from basidiospores. The isolates varied considerably in cultural characteristics, ranging from types with sparse, pale tan, appressed surface mycelium and large, discrete, globular, dark-brown sclerotia, through types with abundant aerial mycelium and loose, amorphous, tan to brown sclerotia, to types with very heavy, cottony, white or tan aerial mycelium and few or no sclerotia.

Cercospora Leaf Spot (Cercospora festucae Hardison = C. apii Fres.). Cercospora leaf spot was found only in trace amounts during the summer of 1949 but was commonly present throughout the State during the summer of 1950. In pastures it appeared to be of little importance. Although as many as 20 percent of the leaves were infected in some pastures, usually there were only a few spots per leaf, and only a trace of damage was recorded. In nurseries at Experiment and Tifton, however, infection on individual plants was much more serious. On some plants 90 percent of the leaves were infected, and many leaves were killed. Estimates of

damage as high as 20 percent were made.

The lesions commonly found on fescue in pastures were small oval to elliptic spots with pale tan centers surrounded by wide dark-brown to purple borders. They measured 0.7-2 x 0.3-0.8 mm. On heavily infected leaves in nurseries such typical eye spots were uncommon. Usually the leaves were heavily marked with irregularly rectangular, dark-brown to purple spots which measured 0.8-7 x 0.2-1.5 mm. For the most part these spots were uniform in color, but occasionally a small area in the center of the spot was pale tan to almost white. Severely infected leaves turned yellow and died back from the tips.

Cercospora festucae did not fruit on spots in living portions of the leaves. When the leaves were placed in a moist chamber for 24 to 48 hours, it produced conidia abundantly in scattered patches on the dead tips. Morphologically the fungus corresponded with the description given by Whitehead and Holt (Phytopath. 40: 1023-1026. 1940). Conidia from Georgia material measured 81-420 x 2.8-5.3 microns (Av. 25= 281.4 x 3.9 microns). Isolates of the fungus were obtained from single conidia and from infected leaf tissue. In culture it produced slow-growing, thick, discshaped, white to greenish-gray colonies. It was difficult to isolate C. festucae from lesions. Colletotrichum graminicola and an Alternaria species of the A. tenuis group appeared most frequently in isolations from leaf tissue. These two fungi also fruited commonly on the dead leaf tips. They probably will prove to be secondary organisms since they fruited as well on leaves dead from any cause and were likewise frequent contaminants in isolations from typical netblotch lesions. Nevertheless, it is apparent that inoculation tests will be necessary to determine whether Cercospora festucae alone is responsible for the symptoms observed.

Spot Blotch (Helminthosporium sativum Pam., King, & Bakke). Spot blotch, which has not previously been reported on fescue, was found in test plots at Dublin in August 1950. It was responsible for only a trace of damage at this location and has not been found elsewhere. The lesions closely resembled those described for Cercospora leaf spot and were distinguished only because Helminthosporium sativum was isolated from the lesions and fruited on the dead leaf tips. The lesions were short to long rectangular, dark-brown spots which were uniform in color or marked with a small buff-colored area in the center. They measured 1-5.5 x 0.5-1 mm. Heavily spotted leaves died back from the tips. The conidiophores and conidia of the fungus were typical of H. sativum as described by Drechsler (Jour. Agr. Res. 34: 641-740. 1923). The conidia measured 67-115 x 16.8-22.4 microns (Av. 25 = 88.9 x 20.0 microns). Isolates from conidia and from the lesions produced a felted, dark-gray to black mycelium with scattered tufts of white hyphae rising above its surface.

White Mold (Sclerotium rolfsii Sacc.). White mold was found on a few plants in a nursery at Experiment and in test plots at Dublin during the summers of 1949-50. It was of infrequent occurrence and therefore of little practical importance. On infected plants heavy mats of the white mycelium of Sclerotium rolfsii grew upward inside of the leaf sheaths and spread out over the base of the blades where it formed the characteristic spherical, brown sclerotia. The leaf sheaths showed a watersoaked, brownish discoloration and later became blackened. Finally the entire plant was blighted, and the leaves turned brown. The shoots at the base of the dead plant were blackened and shredded.

Crown Rot (Various Fungi). Blighted fescue plants were occasionally found at various locations in Georgia in which most of the leaves were dead and the split crowns exhibited a dark-brown to black discoloration of the inner tissues. The discoloration appeared as streaks in the xylem in the upper portion of the crown. In the lower portions it extended throughout the stems, and the bases of the plants were rotted. Blocks of infected stem tissue, cut from the interior of the crown, surface sterilized, and planted on plain water agar, yielded a number of fungi, among which were Helminthosporium dictyoides, Rhizoctonia solani, several Basidiomycetes (Probably Marasmius spp.; M. tritici P. A. Young ? was often found fruiting on dead leaf sheaths), Curvularia sp., Fusarium spp., Trichoderma sp., Thielavia sp., and Aspergillus sp. Inoculation tests will be necessary to determine which of these fungi are primary pathogens. It is possible that crown rot is a disease complex in which several organisms may be involved. R. solani and H. dictyoides probably should be given primary attention since both of these fungi have been isolated by Dr. B. B. Higgins from fescue seedlings killed by damping off in the greenhouse. Marasmius sp. probably should be considered also. At one location this was the only organism, with the exception of three isolates of Trichoderma sp., obtained from a series of 16 tissue plantings.

GEORGIA EXPERIMENT STATION, EXPERIMENT, GEORGIA

AN ASCIGEROUS STAGE OF DARLUCA FILUM (BIV.) CASTAGNE

Paul D. Keener

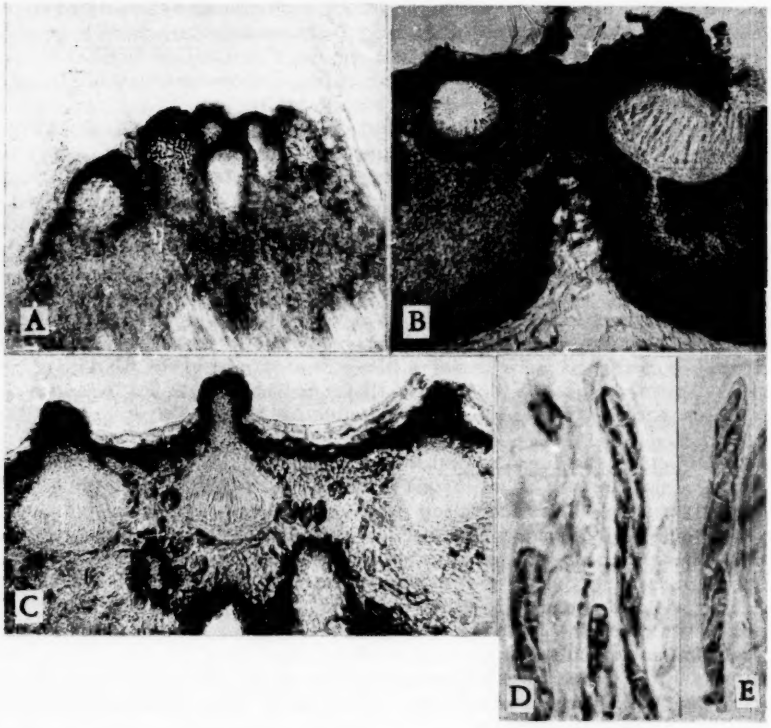

FIGURE 1. Free-hand sections through rust sori showing various features of Darluca and Eudarluca. A. Section through several pycnidia of a typical form of Darluca filum (Biv.) Cast. on Uromyces silphii (Burr.) Arth. Note a few rust spores at the left. B. Section through a stroma of an Ascoymcete believed to represent the Eudarluca phase of Darluca filum on Puccinia extensicola oenotherae (Mont.) Arth. on Carex sp. showing a macropycnidium with macroconidia and a perithecium with asci, in the same general area. C. Section through a stroma of a similar Ascomycete on Puccinia obscura Schroet. on Luzula sp., showing several perithecia with immature asci and other structures (particularly paraphyses). D. Asci and ascospores from the fungus found parasitizing sori of Puccinia extensicola oenotherae on Carex sp. Note the bi-seriate arrangement of the spores. E. Ascus and ascospores from a verified collection of Eudarluca australis Speg. on Puccinia liberta Kern on Eleocharis geniculata R. Br. (tropical collection). A-D. Material from Pennsylvania. E. Material from Dominican Republic. Approximate magnifications: A-C, X 168; D and E, X 825.

Cultural and histological studies at various intervals over several years disclose that a fungus found colonizing uredinial and telial sori of Puccinia extensicola oenotherae (Mont.) Arth., on Carex sp. (= C. straminea ?) at State College, and Puccinia obscura Schroet., on Luzula sp. at Shingletown Gap, Pennsylvania, represents an ascus stage (Eudarluca) of the rust parasite, Darluca filum (Biv.) Castagne (1) (Fig. 1, A). A similar fungus was also observed for the first time in 1933 on Frommea obtusa (Str.) Arth., on Potentilla canadensis L., near State College, Pennsylvania. On P. extensicola oenotherae and on P. obscura, perithecia, as well as asci and ascospores were found in either the same (Fig. 1, B) or in different (Fig. 1, C) stromata in which macropycnidia and macroconidia were produced. Micropycnidia and microconidia were infrequently observed. The arrangement of the ascospores within the asci was either bi- or tri-seriate (Fig. 1, D), which agrees with known collections of certain Eudarluca species (Fig. 1, E) on some tropical rusts. On a few additional rusts, the hyperparasite produced well-developed stromata while on others no stromatic tissues were observed at any season of the year. The presence or

absence of a stroma is apparently independent of the type of rust sorus, the rust spore stage and the phanerogamic host.

Cultures prepared on lima bean agar from single (discharged), as well as groups of ascospores, and from perithecial wall fragments, resulted in the production of typical Darluca filum pycnidia and conidia, along with stromatic tissues common to D. filum in nature on the previously mentioned rusts and on certain others. Results of detailed studies of cultural characteristics of several strains of D. filum, isolated from numerous rusts onto synthetic culture media, have already been presented (2). In addition, the physiological specialization expressed by these isolates in their variable behavior on several species of rusts, was investigated and a report made (3).

The generic name, Eudarluca, was established for the ascigerous stage of Darluca australis by Spegazzini in 1908 (4), citing Eudarluca australis as the type species. The fungus was parasitizing Uredo cannae (= Puccinia cannae (Wint.) P. Henn.). No mention is made of a stroma and the spores illustrated are 3-celled. In 1930, Sydow (5) described Eudarluca venezuelana which he obviously considered to be the ascigerous stage of Darluca venezuelana Syd. nov. spec. (5). In this instance, the fungus was parasitizing a Hyphomycete (? Tuberculariaceae) and the spores were 4-celled. Spegazzini and Sydow apparently never confirmed, by accepted cultural techniques, actual relationships between the Darluca and Eudarluca phases of their respective species.

With the exception of the number of spore septations, the presence or absence of spore appendages and stromata, all of which were found in a previous study to be quite variable in Darluca filum (2, 3), the fungus found in nature in a mature condition on Puccinia extensicola oenotherae on Carex sp. and on P. obscura on Luzula sp., and in an immature state on Frommea obtusa on Potentilla canadensis, agrees morphologically for the most part with the descriptions offered by Spegazzini and Sydow. Since an actual connection between the Eudarluca and Darluca phases was effected by cultural techniques for the rust parasite heretofore commonly recognized as Darluca filum (Biv.) Castagne, during this study, the question is raised as to the necessity for re-characterizing the genus Eudarluca, and for the possible adoption of Eudarluca australis as the proper binomial. A more extensive account of these studies is contemplated for the future.

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A LOCAL, WINTER-TIME EPIDEMIC OF BLISTER SMUT, ENTYLOMA CRASTOPHILUM, ON KENTUCKY BLUEGRASS AT PULLMAN, WASHINGTON

George W. Fischer

It recalls the much-mentioned postman's holiday to find a smut literally in my own back yard, and when it is occurring in epidemic form on New Year's Day it would seem that this unusual situation deserves to be made a matter of record.

The winter weather in the Pacific Northwest so far this season has been as unusually mild as the last two winters have been unusually severe. In eastern Washington, at least, winter wheat has been growing lushly and pastures and lawns have remained green. I had been observing that much of the lawn at my own residence (11 miles west of Pullman) was taking on a definite yellowish cast, quite in contrast to the appearance of other grass in the neighborhood. No particular intellectual curiosity was aroused by this circumstance, however, until I noticed that the etiolated blades of Kentucky bluegrass (Poa pratensis L.) were rather abundantly supplied with blackish spots. Further examination disclosed that these spots were the sori of blister smut, Entyloma crastophilum (E. irregulare Johans., E. speciosum Schroet., E. lineatum (Cooke) Davis, and probably several others).

This does not comprise a new host or State record, but, as nearly as I can determine, this is the first report of the smut in sufficient prevalence and severity as to be causing definite damage to a lawn. The affected portions of my lawn are sickly yellow and each infected leaf is rather abundantly spotted with the small (1/2-2 mm.) blackish sori of the smut fungus. Conidia have not been observed.

In the same lawn, Chewings fescue, (Festuca rubra var. commutata Gaud.), perennial ryegrass (Lolium perenne L.), and annual bluegrass (Poa annua L.) are well represented, but none of the smut could be found on these grasses. In view of the fact that all three have been reported as hosts to Entyloma crastophilum, it would seem that this smut on Kentucky bluegrass may represent a physiologic race to which some of the other hosts of E. crastophilum are resistant.

Besides material for exsiccati distribution, there is an abundant supply of this spot smut on Kentucky bluegrass for teaching purposes. I should be glad to share it with any who request a portion.

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OBSERVATIONS ON BEAN DISEASES IN MICHIGAN DURING 1949 AND 1950

Axel L. Andersen

The problems confronting Michigan bean producers today are similar in many respects to those which confronted them 30 to 40 years ago. Since that time, new navy pea varieties have been introduced and the disease picture is slightly different for these varieties than for older ones. Many of the older varieties are still being grown and in these the disease complex has not changed to any important extent. From two years of field observations, it is apparent that diseases play an important part in lowering the yields and quality of field beans in Michigan. The principal diseases of dry beans in Michigan in 1950 are listed in order of importance in Table 1.

Table 1. Principal diseases of dry beans in Michigan in 1950.

County[a]	Type	Diseases in order of importance
Huron, Bay	Navy pea	Sclerotinia wilt, bacterial blights
Gratiot, Tuscola, and Saginaw	Navy pea	Bacterial blights, sclerotinia wilt
Sanilac and St. Clair	Navy pea	Anthracnose, bacterial blights, rust, and sclerotinia wilt
Montcalm	Navy pea	Bacterial blights
	Cranberry	Anthracnose, bacterial blights, mosaic
	Red kidney	Bacterial blights, anthracnose
	Yellow-eye	Bacterial blight

[a]The principal bean growing counties in the State. Altogether 34 counties are listed as producing dry beans.

For many Michigan farmers dry edible beans are the principal source of income. In 1949, the farmers of this State produced over 90 percent of the pea and medium white, over 90 percent of the cranberry, over 50 percent of the yellow-eye, and approximately 10 percent of the red kidney beans grown in the United States. The total production for all these varieties in the State in 1949 was approximately 5,968,000 one hundred-pound bags valued at $33,000,000.[1]

Although Michigan set a record for the production of dry field beans in 1949, the season was also favorable for the development and spread of bean anthracnose, caused by Colletotrichum lindemuthianum, and of bacterial blights, caused by Xanthomonas phaseoli and X. phaseoli var. fuscans. As a result wherever the pathogens were present, they became distributed uniformly throughout the fields. This was true for the navy pea as well as the colored bean areas. Since the pathogens are seed-borne and a large percentage of the growers either produce their own seed or obtain non-certified locally grown seed from their elevators for the next year's planting, the beans which were planted in 1950 were carrying a considerable amount of bacterial blight infection and, in some sections, of anthracnose. The bacterial blights were common in Tuscola, Saginaw, and Gratiot Counties in 1949, but not in Huron County. Bean anthracnose was present in 1949 in navy beans in Sanilac and St. Clair Counties and in cranberry beans in Montcalm County. In fact, bean anthracnose became so well distributed in cranberry beans in 1949 that it was impossible to obtain clean seed in the State for the 1950 planting season.

The 1950 season was ideal for disease development in beans. As pointed out in the preceding paragraph, the beans were rather generally exposed to the bacterial blight and anthracnose pathogens in 1949. Hence the beans planted in 1950 carried these pathogens in the seed. Heavy rains in July and early August distributed the bacterial blight organisms and anthracnose spores throughout the fields in which inoculum was present. As a result, the beans in Sanilac, St. Clair, and Montcalm Counties were severely damaged and completely defoliated by anthracnose; and the beans in Gratiot, Saginaw, Bay and Tuscola Counties were similarly defoliated by the bacterial blights by the middle of August. The premature defoliation of bean plants probably reduced the yields in these areas by more than 50 percent. Huron County, which had very little bacterial blight infection in 1949, was comparatively free from infection in 1950.

[1]Report of the Michigan Co-operative Crop Reporting Service, December 23, 1949.

Bacterial blights and anthracnose to a certain extent were probably indirectly responsible for additional losses to the bean growers this past season. Wet and humid weather prevailed through the middle of September, making it impossible for the growers to harvest their beans. As a result, the prematurely ripened beans were severely damaged by weathering (molding and sprouting of the beans in the pods). This damage increased the dockage (pick) by 25 to 30 percent. Direct evidence was available to indicate that the above supposition was true. A field of beans in Tuscola County belonging to the Foundation Stock Seed Association was free from bacterial blights and other diseases. This field was planted on June 1, and on September 12 it was just beginning to mature normally. Some of the pods were green, most of them were yellow, and a few were ripe. The yield from this field was 35 bushels per acre. During the week of September 10 to 16, severe weather damage occurred on the prematurely ripened beans standing in the fields. This particular field was only slightly injured by weather. Had the other fields in the county and in Saginaw and Gratiot Counties been free from bacterial blights, they, too, would have escaped much of the injury from the weather. Tuscola, Saginaw, and Gratiot Counties produced in 1949, approximately 2,094,000 100-pound bags of navy pea beans. In 1950 the total production for these three counties will probably be reduced 60 to 70 percent. Assuming that 30 percent of the reduction was due to drowning and reduction in acreage, then 30 to 40 percent of the reduction in total yield is directly or indirectly due to bacterial blights. This is a 500,000-bag or $3,500,000 loss to the growers in these three counties. The actual losses to the producer are greater because he is docked 15¢ per pound of pick in the beans he sells to the elevator.

A similar story as regards been anthracnose can be told for Sanilac and St. Clair Counties. Approximately 60 percent of the bean fields visited in these counties were affected with anthracnose. The direct reduction in yield due to killing of the leaves and plants in the infected fields was estimated at 50 percent. Further reductions would result from the increase in pick due to infected seeds and weathering, a factor which is associated with the premature killing of the plants due to defoliation. These two counties produced 840,000 100-pound bags of navy pea beans in 1949. Allowing for a 20 percent acreage reduction and assuming a 50 percent reduction in yield on 60 percent of the remaining acreage and not taking into account the increase in pick due to infected seeds and weathering, an estimated loss of 200,000 100-pound bags or $1,400,000 can be attributed directly to bean anthracnose.

Although the beans in Huron County were comparatively free from bacterial blights and anthracnose, a considerable proportion of the fields were severely injured by Sclerotinia sclerotiorum, which causes Sclerotinia wilt, or watery soft rot. Many fields in which 50 to 90 percent of the beans had been killed by this fungus were observed. The disease was present in fields which had never grown beans and in those 10 years removed from beans. The fields which suffered the greatest losses were those in which alfalfa or clover had been turned under prior to the planting of beans, or those which had been heavily fertilized. In general, all the bean fields which had heavy vine growth were severely injured by S. sclerotiorum. A conservative estimate for Huron County would be 5 to 10 percent of the total crop lost because of infection with this fungus or approximately 7,000 100-pound bags valued at $50,000.

Several diseases of minor importance were noted throughout the State during the past two years. These include the root rots, rust, powdery mildew, and mosaics. Root rot was severe in isolated fields throughout the States; the exact identity of the causal pathogens is not known. Occasional outbreaks of bean rust (Uromyces phaseoli var. typica) and powdery mildew (Erysiphe polygoni) were noted. In such cases these diseases had completely defoliated the plants. Bean virus diseases were prevalent in some fields in 1949, but insignificant in 1950.

In comparison with the dry bean industry, the growing of garden beans for canning is a relatively small industry in this State. Two diseases of garden beans have been of commercial importance during the past two years. Pythium wilt, caused by Pythium butleri, was present in many fields, and it caused stand reductions as high as 20 to 30 percent. Several fields of garden beans in Montcalm County had to be abandoned this year because of bean anthracnose. The beans observed in Montcalm County were all of the variety Topcrop, a new variety on the market last year.

DIVISION OF FRUIT AND VEGETABLE CROPS AND DISEASES, BUREAU OF PLANT INDUSTRY, SOILS, AND AGRICULTURAL ENGINEERING, AGRICULTURAL RESEARCH ADMINISTRATION, U. S. DEPARTMENT OF AGRICULTURE AND DEPARTMENT OF BOTANY AND PLANT PATHOLOGY, MICHIGAN STATE COLLEGE, EAST LANSING, MICHIGAN

NOTEWORTHY DISEASES OF CUCURBITS IN NORTH CAROLINA IN 1949 AND 1950

D. E. Ellis

Rhizoctonia Fruit Rot of Cucumber. In July 1949 Earliest of All variety cucumbers (Cucumis sativus L.) from Pitt County were affected with a rather firm, dark, water-soaked decay extending well into the flesh. Affected cucumbers held in moist chambers at room temperatures (25-31° C.) rotted rapidly and after 24 hours were covered with profuse aerial mycelium of a fungus believed to be Rhizoctonia solani Kühn. This aerial mycelium was almost white and developed so abundantly on fruits in the moist chambers that in some respects it resembled cottony-leak caused by Pythium spp. The water-soaked type of decay was common on the larger fruits (over three inches long) while on smaller, No. 1 size fruits a yellowish-brown superficial discoloration was noted. Microscopic examination revealed that the tissues of the former were gorged with Rhizoctonia mycelium. This fungus was consistently isolated from affected fruits in this and several collections subsequently obtained from the same area. On the smaller fruits showing the yellowish-brown discoloration, Rhizcotonia myeclium was present on the surface but had not penetrated deeply into the flesh.

Nine single-hyphal-tip-isolates of R. solani were secured from affected fruits and all proved highly pathogenic to healthy National Pickling variety cucumbers in laboratory inoculation tests. These were surface disinfected with sodium hypochlorite solution, placed in moist chambers, and inoculated with potato-dextrose-agar cultures of the fungus. At room temperature (25-31° C.), first evidence of decay was noted within 24 hours. It progressed rapidly so that after 72 hours entire fruits were almost completely rotted. After five days the cucumbers were completely broken down into a watery mass of tissues covered with a profuse growth of mycelium and sclerotia. The decay progressed equally rapidly in wounded and non-wounded cucumbers. Controls, to which sterile agar was added, remained sound. In greenhouse inoculation tests the fungus caused severe damping-off of cucumber seedlings.

While the causal fungus conforms to R. solani morphologically, it grows more rapidly than several isolates of that fungus from other hosts with which it was compared. At 28° C. it covers the medium in a 90 mm. Petri dish within 48 hours. Preliminary tests indicate that the optimum temperature, both for growth of the fungus and disease development, is 28° C. or above. In preliminary tests, the isolates were found to infect tomato and pepper fruits, causing symptoms resembling soil-rot on these hosts. While R. solani causes damping-off and stem rot of cucumber (4) it apparently has not previously been reported as a cause of fruit-rot of that host.

Squash Scab and Powdery Mildew. Scab (Cladosporium cucumerinum Ell. & Arth.) has for several years been a limiting factor in the production of summer squash (Cucurbita pepo L. var. condensa Bailey) in the mountain area of North Carolina. While the disease has not been particularly troublesome in early plantings harvested in mid-summer, it has been very serious in late plantings which are often desirable because of the usually higher market prices that prevail late in the season. In 1949 several late plantings in Henderson County were total losses due primarily to scab. While C. cucumerinum has been widely reported on cucumber, its occurrence on squash has previously been recorded in the United States only from Maryland, Massachusetts, New York and Pennsylvania (4).

While fungicides have been relatively ineffective against scab on cucumber, some have shown promise (1). It therefore seemed desirable to explore the possibilities of controlling the disease on squash with frequent applications of fungicides. Results of a preliminary fungicidal spray test at the Mountain Vegetable and Fruit Station, Hendersonville, in 1950, are given in Table 1. While scab was not so severe as in 1949 it developed rather abundantly late in the season, as also did powdery mildew (Erysiphe cichoracearum DC). Of the several fungicides included, only zineb showed much promise against scab. Although certain other materials appeared to reduce scab incidence, on a percentage basis, zineb was the only material that resulted in significant yield increases. Furthermore, for the last three pickings, when scab was most severe, the percentage of scabbed fruit on zineb-treated plots was significantly less than for any other treatment. Cycloheximide, which has shown some promise against scab of cucumber in Michigan (1), was relatively ineffective against squash scab in this test. However, it was very effective against powdery mildew. Cycloheximide has previously been found effective against bean powdery mildew (E. polygoni DC) (3). Cuprous oxide plus cotton-seed-oil was also apparently very effective against the squash powdery mildew. However, this treatment was noticeably phytotoxic.

Table 1. Effect of fungicides upon yield and incidence of scab and powdery mildew of summer squash. [a]

Treatment [b] Materials and concentration	Yields of marketable squash (bushels per acre)		Scabbed fruit (percent)		Powdery mildew [c] (Index of severity)
	Entire season	last 3 pickings	Entire season	last 3 pickings	10/3/50
Cuprocide 1-100 + cotton-seed-oil (1 gal./100 gal.)	267	56	8.2**	21.8*	0.0
Cotton-seed-oil 1 gal./100 gal.	281	67	15.5	28.9	1.5
Actidione 10 p.p.m.	286	52	12.6	30.7	0.0
Tribasic copper sulfate 2-100	291	56	7.8**	20.0*	0.7
Crag Potato Fungicide 658 2-100	323	65	11.2*	33.7	0.7
Parzate 2-100	390**	95**	1.9**	5.0**	0.7
Nontreated check	277	53	18.7	35.8	2.5
L.S.D. 0.05	58.1	20.5	7.0	12.3	-
L.S.D. 0.01	79.6	28.1	9.6	16.9	-

[a] The assistance of C. E. Lewis, R. G. Owens and H. C. Fink is gratefully acknowledged.

[b] Four replicate plots, 0.007 acre each, were used for each treatment. Seeds of Early Prolific Straightneck squash were planted on July 20, 1950 and seedlings later thinned to 12 plants per plot. The sprays were applied with 3-gallon knapsack sprayers at rates of approximately 100 gallons per acre per application, on the following dates: Aug. 24, 28; Sept. 2, 6, 11, 15, 19, 22, 27; Oct. 3, 7, 1950. Scab infection was first noted on both leaves and fruits late in August and developed slowly until mid-September when rains favored its more rapid development and spread.

The following materials were supplied by the Manufacturers: Cuprocide (83% cuprous oxide) by Rohm and Haas Co.; Actidione (cycloheximide) by the Upjohn Co.; tribasic copper sulphate (53% copper) by Tennessee Corp.; Crag Potato Fungicide 658 (copper zinc chromate - 30% copper, 20% zinc, 10% chromium) by Carbide and Carbon chemicals Corp.; Parzate (65% zineb) by E. I. DuPont de Nemours Co., Inc.

[c] 0= no mildew; 1+ slight; 2= moderate; and 3= severe mildew infection.

* Significantly different from nontreated check.

** Highly significantly different from nontreated check.

Cucurbit Downy Mildew. As previously pointed out (Plant Disease Survey Warning Service Report No. 42. 1950), downy mildew (Peronoplasmopara cubensis (Berk & Curt.) Clinton) (Pseudoperonospora cubensis (Berk. & Curt.) Rostow.) developed more slowly on cucumber than it did on muskmelon (Cucumis melo L.) in 1950. This led to the speculation that there might be two or more physiological races of the fungus and that in 1950 we were dealing with a race different from that most prevalent in North Carolina during the past several years. At the Coastal Plains Vegetable Station, Faison, the disease developed slowly on susceptible cucumber varieties, which continued to yield for a considerable time after susceptible muskmelon varieties had been killed. The same situation prevailed in commercial plantings, particularly in the Ridgeway area where the disease caused much more damage to muskmelon than to cucumber. The behavior of the disease in 1950 was in marked contrast to the more normal situation in which it usually is first noted and develops most rapidly on cucumber; appearing on muskmelon from a week to ten days later, and on watermelon (Citrullus vulgaris Schrad.), still later than on muskmelon. This so-called "normal" behavior also suggests the possibility of the occurrence of races which differ in their virulence on the various cucurbit hosts.

Reports from Florida indicated that the disease developed slowly on cucumber owing in part to dry weather, but later caused considerable damage to watermelon. Reports from several other areas indicated that the disease was severe on watermelon, and in Virginia (Warning Service Report No. 46) and Delaware (Report No. 49), the disease was unusually severe on muskmelon.

Studies in South Carolina (2) have shown that while the downy-mildew resistant Palmetto variety of cucumber may show infection, especially when interplanted with susceptible varieties, the fungus seldom sporulates on it. In North Carolina in 1950, fall plantings of the Palmetto

variety in Duplin and Pender Counties were rather seriously affected by downy mildew. Where fungicides were not used, the disease caused considerable defoliation, even where no susceptible varieties were interplanted. While the fungus did not sporulate as profusely on the Palmetto variety as it usually does on more susceptible varieties, rather abundant sporulation did occur on it and an occasional Palmetto leaf was observed on which sporulation was so heavy that the lower surface was almost black. This further suggests the possibility that physiological races may exist.

Whether or not there are races of P. cubensis that differ in their pathogenicity is of course a matter of speculation at present. However, it is a question that needs to be answered and will require careful work under controlled conditions. One serious limitation to such a study would be the necessity of maintaining cultures of the fungus on living plants. It is questionable whether this could be done in areas where the disease does not normally over-winter without encountering a danger of starting an abnormally early epiphytotic of the disease in the spring.

Other Diseases. Gummy stem blight, caused by Mycosphaerella citrullina (C.O. Sm.) Gross., appears to be increasing in importance. It has been noted on watermelon in many eastern counties for the past several years and in 1949 caused very severe defoliation in a few plantings in Wake and Warren Counties. The fungus was found associated with blossom-end-rot of watermelon in Moore and Scotland Counties in 1949 and in Wake County in 1950. It also caused a leaf spot of muskmelon in Scotland, Wake and Warren Counties in 1949 and 1950.

Anthracnose (Colletotrichum lagenarium (Pass.) Ell. & Halst.) caused as much, or more, damage to cucumber in 1950 as did downy mildew. It was especially serious on pickling varieties where the seed were not treated with mercury bichloride solution. The disease causes important losses of watermelons each year. In test plantings the new Congo variety has been highly resistant.

Bacterial wilt (Erwinia tracheiphila (E.F. Sm.) Holland) of cucumber, which is usually of minor importance, caused losses of from 2 to 20 percent in Chowan and Pasquotank County plantings in 1949.

Mosaic (virus) was unusually severe in experimental plantings of cucumber and summer squash in Duplin County in the fall of 1950.

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CONTROL OF SMUT IN BULB ONIONS BY PELLETING THE SEED WITH TECHNICAL THIRAM

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In Cook County, Illinois, the pelleting of bulb-onion seed with Arasan (50% thiram, non-wettable in water) has replaced almost entirely the formaldehyde-drip treatment as a control for smut. The standard dosage and treatment, as worked out originally by Newhall (2) and still used in this area, is one pound of fungicide to one pound of seed which is moistened prior to pelleting with a 5 percent Methocel (Dow methyl cellulose) sticker solution. [1] That this is a fairly simple treatment is evidenced by the fact that practically all onion-seed pelleting in Illinois is done by the individual grower. Many growers use a rotating, wooden butter churn to mix seed and fungicide, treating five or more pounds at a time.

The present method of treatment leaves much to be desired because of the necessity of holding relatively heavy dosages of fungicide on the seed. Arasan does not stick to the seed as well as Tersan (50% thiram, wettable) and tends to cause more "bridging" of the seed in the drill box. However, it has given appreciably better smut control than Tersan and control comparable to formaldehyde in experiments by the writer and comparative tests by growers. For these reasons, Arasan is used more widely than Tersan by Cook County growers despite the fact that Tersan was the first dust fungicide used on a commercial scale for smut control in the county. Difficulties in obtaining an even flow of seed from the drill have been overcome to some extent by stirring the Arasan-pelleted seed in the hopper with a wooden paddle at infrequent intervals. It should be pointed out here that Illinois onion growers have no particular interest in any method faintly resembling the formaldehyde-drip treatment, wherein a fungicide is added separately to the soil (3, 4).

This paper is a report of experiments conducted in 1950 and designed to find ways to reduce the fungicide-seed dosage ratio and at the same time to maintain the concentration of thiram. The results are given in the Reporter with the hope that other interested pathologists can try technical thiram for smut control in 1951.

MATERIALS AND METHODS: Various concentrations of thiram were tested to determine their effect on onion seed emergence in flats, on the rate of seeding and on smut control in the field. In addition to Arasan and Tersan, these included technical thiram [2] and Arasan SF (75% thiram, wettable). The fungicides were stuck to the seed with a 5% solution of 15 centipoise Methocel containing 10 percent glycerin. The quantity of sticker used, which is given in Table 1, was determined on the basis of the dosage level and comparative wettability of the fungicide.

Table 1. The effect of various concentrations of thiram on emergence of old and new onion seed.

Fungicide	Percentage thiram	Dosage level on seed (%)	Amount of Methocel-glycerin solution (%)	Percent emergence: 1946 seed, Test 1, Soil	1946 seed, Test 2, Soil	1946 seed, Test 2, Sand	1949 seed, Test 1, Soil	1949 seed, Test 1, Sand
Thiram	100	100	50 [a]	19.0	21.6	26.6	76.6	85.0
"	"	75	25 [b]	18.0	-	-	88.3	93.3
"	"	50	20	25.0	-	-	95.0	98.3
Arasan	50	100	50	11.5	20.0	3.3	63.3	45.0
Tersan	50	"	40	-	-	-	86.6	85.0
Arasan SF	75	"	40	17.0	51.6	26.6	70.0	76.6
" "	"	75	25	27.5	33.3	43.3	96.6	86.6
Check	-	-	-	68.0	76.6	60.0	98.3	93.3

[a] Originally set at 30 percent; but increased through error.
[b] Roughly equivalent to 1 pint to 4 lbs. of seed which is the rate usually recommended.

[1] Tests in Illinois show that the addition of commercial glycerin (10 percent by volume) as a plasticizer (1) reduces shattering or flaking-off of the fungicide, presumably by making the Methocel film less brittle or drying.

[2] The Monsanto Chemical Company, St. Louis, Missouri, kindly supplied a finely-ground grade of technical thiram for these tests.

The seed in a one-liter flask was moistened with the sticker and stirred with a narrow wooden paddle until each seed was completely wetted. Following this the required amount of fungicide was added and the whole stirred with the paddle for two minutes. The flask was then shaken and rolled until the pellets separated. The seed-fungicide mass was stirred for one-half minute with the paddle to break up any seed clumps and again shaken until all the fungicide had been taken up by the seed and formed into pellets. The treated seed was allowed to dry at 65° to 75° F. for at least 24 hours prior to sowing.

Emergence tests were run on two different lots of seed -- harvested in 1946 and in 1949 -- sown in soil and in sand flats. Sixty seeds from each treatment were planted in each non-replicated row. The 1949 seed was placed in furrows lined with "fibre-spun" paper so that the near-maximum phytotoxic effect of the fungicide would be obtained. The soil and sand, which had been steam-pasteurized, were kept moderately wet but not saturated and at temperatures of 55° to 57° F. after seeding. Stand counts were made at approximately three-day intervals over a period of four weeks after initial emergence.

Pelleted seed was sown in the field on April 21 using a Model No. 300 Planet Jr. drill with each treatment replicated four times. Plots of 200 seedlings each were staked out on May 25 when the majority of the plants were from two to three inches tall. Smut-affected seedlings were noted at this time but no dead plants were evident until three weeks later. Counts on apparently smut-free plants were made on June 22 and August 2, and final data on smut-free bulbs at harvest on September 8.

Table 2. The effect of various concentrations of thiram on rate of seeding and control of smut in bulb onions. (Seeder plate hole No. 8 used for pelleted seed, No. 7 for check).

Fungicide	Percentage thiram	Dosage level on seed (%)	Pounds of actual thiram per acre[a]	Mean no. feet of row - 200 plants	Percentage smut: June 22	August 2	September 8 (harvest)
Thiram	100	100	4	72	0.5	13.5	19.6
"	"	75	3	49	3.9	14.9	21.5
"	"	50	2	52	5.6	19.7	30.5
Arasan	50	100	2	45	8.5	30.5	45.7
Tersan	50	"	2	43	7.9	33.2	46.7
Arasan SF	75	"	3	44	8.1	34.9	43.4
" "	"	75	2.2	41	9.2	40.5	48.6
Check	-	-	-	38	39.6	69.9	85.4
Least difference necessary for significance (5%) level)					6.3	7.9	6.8

[a]Based on a seeding rate of 4 lbs. per acre.

DISCUSSION OF RESULTS: The data in Tables 1 and 2 show that technical thiram (1) was somewhat less phytotoxic at the two lower dosage levels than Arasan regardless of the age of the seed, (2) gave significantly better smut control than Arasan, Arasan SF and Tersan at all concentrations, and (3) did not interfere appreciably with the rate of seeding except at the highest concentration (See "Mean no. of feet of row - 200 plants" in Table 2).

The pellets resulting from treatment with technical thiram at 50 and 75 percent dosage levels were smoother and harder than those incorporating Arasan or Tersan. The slight interference with seeding experienced at these concentrations and presumably due to interfacial friction among the pellets could have been compensated for by using a larger plate-hole size. The plant spacings were approximately the same in all treatments with the exception of thiram at 100 percent dosage. Through an error an excessive quantity of sticker was employed at this concentration, resulting in clumping of the pellets. The amount of sticker used appears to be rather critical, since too much can cause aggregation of seed and too little often results in loss of the fungicide from the seed. For example, in tests preliminary to those reported here it was found that 25 percent sticker -- the quantity generally recommended for Tersan and Arasan -- allowed as much as one-fourth of the Arasan to shatter off, whereas 50 percent sticker permitted a loss of less than 5 percent.

Although smut control with the best treatments may appear to have been rather poor, the results on the whole were good considering the weather conditions, which were ideal for the smut fungus. During the three-week period following seeding, rainfall totaled 3.9 inches and the daily

mean temperature range was between 38° and 57° F. The seedlings were slow in emerging and as a result were exposed to smut infection for a comparatively long period. Another factor not to be overlooked is the high smut potential in this field, [3] which would make it impossible to obtain near-perfect control with treatments now available. From 95 to 100 percent of the plants from untreated seed are killed in many Cook County fields by smut or have the disease in the bulbs at harvest.

Technical thiram will be used on a limited commercial scale in Cook County in 1951 if a finely-milled grade is available.

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[3] Percentage smut in this same location in 1944 was: Arasan (1:1) 7.7; formaldehyde-drip 8.4; and check 85.2.

ROOT KNOT SUSCEPTIBILITY OF LYCOPERSICON PERUVIANUM

A. L. Taylor and B. G. Chitwood[1]

Only a part of the Lycopersicon peruvianum (L.) Mill. plants tested by Ellis (2) were reported as susceptible to root-knot nematodes, showing a few small galls when planted in a field where the majority of plants of three commercial varieties of the common tomato, L. esculentum Mill. were heavily infected.

Recent investigations of the host relationships of various species of root-knot nematodes make it possible to amplify this report and to suggest a possible explanation of the small amount of infection found in L. peruvianum. In greenhouse trials with single species inoculum at the Division of Nematology, Beltsville, Maryland, L. peruvianum was not infected by populations of Meloidogyne incognita (Kofoid & White) from several sources. It was heavily infected by a population of M. arenaria (Neal) similar to that described by Chitwood (1) from Boehmeria utilis Hort. It was moderately infected by a population of M. incognita var. acrita Chitwood, obtained from Tifton, Georgia, and by a population of M. hapla Chitwood from a spiraea plant collected at Wentzville, Missouri. In all cases the infection appeared to be normal.

M. incognita is reported by Chitwood (1) to be the most common root-knot nematode in the southern United States and various subtropical American Islands. The other root-knot nematode species tested are less generally distributed in the United States, but have been found in various parts of the country and are by no means rare.

It seems possible that the field used by Ellis (2) for his tests had as its dominant root-knot nematode species M. incognita, but that at least one other species was also present in small numbers, and that the infection found on L. peruvianum was due to these latter nematodes.

As Watts (3) has pointed out L. peruvianum is a possible source of nematode resistance in the development of commercial tomato varieties, and since M. incognita is the most common root-knot nematode species in a large part of the country, tomato varieties having resistance to this species would be of great value. But it would also be probable that such varieties would fail to exhibit root-knot resistance where some other species of Meloidogyne are present.

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[1]Division of Nematology and formerly Division of Nematology respectively, Bureau of Plant Industry, Soils and Agricultural Engineering.

TESTS OF FUNGICIDES FOR THE CONTROL OF TOMATO PLANT DISEASES IN SOUTH GEORGIA, 1949-1950

Huey I. Borders

Fungicide tests for control of infections of tomato seedlings caused by Alternaria solani (Ell. & G. Mart.) Sor. have been conducted for several years at the Georgia Coastal Plain Experiment Station, Tifton, Georgia. However, in recent years there has not been sufficient Alternaria infection during the regular spring transplant-growing season to yield clear-cut differences in control by the various materials tested.

For this reason it was decided to conduct additional fungicide tests during the summer months when it was expected that tomato plants would be subjected to more severe attacks by Alternaria leaf spot and stem canker than are ordinarily encountered during the regular spring transplant-growing season.

SUMMER FUNGICIDE TESTS, 1949: Previous tests had shown that the extreme summer heat in south Georgia caused uneveness of stands of seedlings from field-sown tomato seed. It was therefore decided to set out small transplants which would have a better chance of becoming established before the onset of the high summer temperatures. This method was found to be very satisfactory and was followed in the summer fungicide tests subsequently described.

Because of the exploratory nature of the first summer test only a small area was prepared for the plots and the treatments were limited to four sprays. No dusts were used. Transplants of the Rutgers variety were set out on June 7, 1949, and treatments were replicated four times in randomized blocks. The sprays were applied at seven-day intervals from June 28 to August 9 with a power sprayer at approximately 400 pounds pressure.

No Alternaria blight or any other diseases were observed until the last half of July, when a few lesions of Alternaria appeared in the check (unsprayed) plots and in one of the sprayed plots. By the first week in August the plants in the check plots were severely infected and the differences in protection afforded by the various materials were beginning to show. Alternaria spread rapidly throughout the plots from this time on, and by August 11 the check plots were almost completely "burned up" by the disease.

On that date the leaflets infected by Alternaria and the lesions on stems and petioles were counted. The results of these counts are shown in Table 1.

As shown in Table 1 the materials can be grouped according to their effectiveness in controlling Alternaria blight. Disease in the plots sprayed with any one of the four materials tested was significantly less than in the checks. There are also significant differences between the sprays in the group giving the best control, Nos. 1 and 2, and those in the second groups, Nos. 3 and 4. It should be pointed out that the differences between the materials in the first group, and those in the second group were greally greater than appear in the Table. The reason for this is that in making the counts of infected leaflets a leaflet having one small Alternaria lesion was counted as one and the same value was given to a leaflet covered with Alternaria lesions. The foliage of the plants in the plots sprayed with materials Nos. 1 and 2 was so free of disease that a minute inspection of both surfaces of eacn leaflet was necessary in order to find such Alternaria lesions as existed. On the other hand, the disease lesions on the leaflets of the plants treated with materials Nos. 3 and 4 and in the checks were so numerous that counting the infected leaflets required but a glance at each leaflet.

The only insect problem encountered was an infestation of Colorado potato beetle (Leptinotarsa decemlineata (Say)), which was controlled by the addition of calcium arsenate to the sprays. This particular insecticide was used because it is the one most generally employed by the plant industry. It is compatible with the spray materials to which it was added and there was no evidence of deleterious effects from its use.

SUMMER FUNGICIDE TESTS, 1950: The fungicide tests of the summer of 1950 were conducted with the same variety and in the same manner as those of the summer of 1949, but on a larger scale. Both sprays and dusts were tested. Applications were made at seven-day intervals from June 13 to July 25, 1950.

The plots were replicated in four randomized blocks. Each test spray plot consisted of three plant rows 52 inches apart with the plants set two feet apart in the row. There was a total of 30 plants in each spray plot replicate. As it is difficult to control drift in applying dusts the individual dust plots were somewhat wider than the spray plots and comprised four plant rows of five plants each, set two feet apart in the row.

Table 1. Tomato fungicide test, summer 1949.

Treatment -- Material and amount used per 100 gallons [a]		Number leaflets infected		Percent of leaflets infected	Number stem and petiole lesions		Degree of infection[b]
		Total	Mean	Mean	Total	Mean	
1--							
Nabam (Dithane D-14)	2 qts.						
Zinc sulfate	1 lb.	143	36	6.04	6	2	.5 to 1
Lime	1/2 lb.						
2--							
Ziram (Zerlate)	2 lbs.	152	38	6.52	17	4	.5 to 1
3--							
Tribasic copper sulfate	4 lbs.	1662	416	62.20	128	32	3.5
4--							
Phygon XL	1 lb.	1886	472	58.92	203	51	3
5--							
Check		3534	884	97.87	1124	281	5
L.S.D. @ .05 level			131.62	11.93		60.02	
L.S.D. @ .01 level			184.54	16.73		84.16	

[a]Calcium arsenate was used as the insecticide at 4 pounds per 100 gallons in all sprays and on check. Rate of application: 125-150 gallons of spray per acre.

[b]Degree of infection of plants by Alternaria solani based on a scale of 0, 1, 2, 3, 4, 5 in which 0 means complete control and 5 indicates severe infection of plants.

Transplants were set out in the plots on June 6, 1950, and grew well. A few plants were reset to replace transplants killed by soil-borne fungi and bacteria, but these quickly became established and were soon indistinguishable from the original transplants.

Although a few Alternaria lesions were found in some of the plots on June 26, the spread of the disease was slow and the attack did not become heavy until the middle of July. However, the disease steadily increased until the checks and the poorer fungicide plots were almost completely destroyed by the time the test was terminated on August 1, 1950.

Calcium arsenate at 4 pounds per 100 gallons served to control the relatively light outbreak of Colorado potato beetle, which constituted the only insect problem encountered during this test.

The data from this test were recorded and tabulated in the same manner as those from the 1949 test (Table 2). Data on leaf spotting were based on counts made of lesions on 15 compound leaves taken from each of the six middle plants of each plot. Statistical analysis of these data shows that there were highly significant differences in the effectiveness of many of the materials used.

Although on a larger scale than the tests of 1949, these experiments yielded comparable results and the results fall into the same pattern of control afforded by the various materials used in 1949.

It should be pointed out that these experiments were conducted in the presence of far more disease than is ordinarily encountered during the regular tomato transplant growing season. The differences in disease control under these severe conditions were brought out and materials that afforded mediocre control when the Alternaria attacks were light to moderate were shown to be unsuitable under the severe disease conditions.

The results of these experiments indicate that under even extremely severe conditions there are available to the tomato plant grower several fungicidal materials which will yield a relatively high degree of control of Alternaria blight. In all cases disease control was much better from the fungicide, in spray form, as compared with the same materials in dust form.

Another point that should be emphasized is that of the four best spray materials viz., nabam (Dithane D-14) plus zinc sulfate and lime, ziram (Zerlate), zineb (Dithane Z-78), and tribasic

Table 2. All spray and dust tests for control of tomato plant diseases, summer 1950.

Treatment	Number leaflets infected		Percent of leaflets infected	Number stem and petiole lesions		Degree of infection[c]
	Total	Mean	Mean	Total	Mean	
SPRAYS						
Material and amount used per 100 gallons [a]						
1-- Nabam (Dithane D-14) 2 qts. Zinc sulfate 1 lb. Lime 1/2 lb.	431	108	13	28	7	.5 to 1
2-- Ziram (Zerlate) 2 lbs.	991	248	28	41	10	1 to 1.5
3-- Zineb (Dithane Z-78) 2 lbs.	1238	310	32	129	32	1 to 2
4-- Tribasic copper sulfate 4 lbs.	1526	382	41	111	28	2
5-- Copper Cpd "A" 4 lbs.	2045	511	48	177	44	3
6-- Zineb (Parzate) 2 lbs.	3549	887	80	1591	398	4
7- Phygon XL 1 lb.	3979	995	87	2664	666	5
DUSTS [b]						
8-- Ziram (Zerlate) 4131 10-20-70	4131	1033	99	1609	402	5
9-- Zineb (Dithane Z-78) 10-20-70	4243	1061	96	2637	659	5
10-- Tribasic copper sulfate 14-20-66	4345	1086	97	1562	391	5
11-- Phygon XL 10-20-70	4464	1116	100	5901	1475	5
12-- Check 0-20-80	4773	1193	100	10,995	2749	5
L.S.D. @ .05 level		142.56	10.93		439.06	
L.S.D. @ .01 level		191.30	14.66		589.16	

[a] Calcium arsenate was used as the insecticide at 4 pounds per 100 gallons in all sprays and on check. Rate of application: 125-150 gallons of spray per acre.

[b] Dusts: formula represents percent by weight of fungicide, insecticide, and filler. Rate of application: 30 pounds per acre.

[c] Degree of infection of plants by *Alternaria solani* based on a scale of 0, 1, 2, 3, 4, 5, in which 0 means complete control and 5 indicates severe infection of plants.

copper sulfate, two, i.e., nabam (Dithane D-14) plus zinc sulfate and lime, and zineb (Dithane Z-78), also afford a high degree of control not only of late blight, caused by Phytophthora infestans (Mont.) DBy., but also of Stemphylium or gray leaf spot, caused by Stemphylium solani Weber. The efficacy of these two materials in the control of late blight has been reported by numerous workers and their effectiveness in the control of Stemphylium or gray leaf spot of tomatoes has been reported recently by Conover (1).

Late blight has appeared in the Georgia plant-growing area only at intervals so far, and gray leaf spot of tomatoes is as yet rare in its appearances in plant fields in Georgia. Nevertheless both these disease constitute an everpresent threat to the plant grower and when they do appear, the damage caused may be sudden and extensive. For this reason a fungicide which will protect the plants from these diseases as well as from Alternaria blight is required.

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FOUR VIRUS DISEASES OF SOLANACEOUS PLANTS IN TRINIDAD

I. A. C. Ferguson[1]

Symptoms of virus infection are common on Solanaceous plants in Trinidad, British West Indies. In particular, tomato (Lycopersicon esculentum), eggplant (Solanum melongena), sweet pepper (Capsicum annuum), and tobacco (Nicotiana tabacum), are affected.

The presence of the tobacco mosaic virus disease on the Island has already received some attention (1). Other virus diseases described for the first time in this paper have been tentatively named: tomato twisted leaf, tomato bronze leaf, eggplant mosaic, and pepper mosaic.

DISEASES, SYMPTOMS AND HOST RANGE

Table 1 gives a comparison of the symptoms produced in various hosts by the viruses of these diseases.

1. TOMATO TWISTED LEAF: This disease commonly appears in a crop shortly after transplanting, the first signs being a marked check to growth, a shortening of the internodes and petioles, and a downward twisting of the leaves. Leaflets are reduced in size, and those which are expanding at this stage may show marked, yellow vein-clearing. In the variety Ponderosa, necrosis has also been observed along the veins, but the other varieties which were investigated (Marglobe, Winsall, Bonny Best, and hybrids of tomato and Lycopersicon pimpinellifolium) showed only slight veinal necrosis. After several days, infected plants appear to recover and normal height may be attained, but the leaves remain smaller than those of healthy plants and show an interveinal chlorosis. The symptoms of the disease vary considerably, even on individuals of the same variety. In the greenhouse, stunting and twisting of affected plants are suppressed and the most striking symptom under these conditions is the chlorotic pattern on the leaves. From observations on infected plots, the diseased plants bear fewer and slightly smaller fruits.

The disease can not be transmitted by rubbing, but is readily transmissible by grafting. First symptoms appeared within one to two weeks after grafting. By this means the infection was transmitted to sweet pepper (Capsicum annuum), which showed a variegated effect, consisting of ill-defined dark and light patches on the leaves six days after grafting; there was also some yellowing along the veins, and after ten days the leaves became puckered and in some cases the dark green patches became raised to give a blistered appearance. These symptoms are quite distinct from those produced on this host by the other viruses described below. On Solanum melongena, Nicotiana glutinosa, and N. tabacum no symptoms were produced by the virus causing this disease.

2. TOMATO BRONZE LEAF: Tomato plants grown in greenhouses for experimental purposes frequently become infected with bronze leaf and occasional outbreaks have also been observed in the field. The first symptom as observed on the variety Marglobe is a slight bronzing of the young leaves, which soon becomes marked. These leaves remain small and develop a leathery texture. The growth of the plant is severely retarded and a necrosis accompanied by a dieback of the growing points sometimes develops, resulting finally in the death of the plant. Fruits from infected plants frequently show necrotic patches on the epidermis, or may show no symptoms at all. The disease has been readily transmitted by grafting and by rubbing where the expressed virus sap was immediately rubbed on to the host. When the expressed sap was exposed to the air for an hour infections of less than 1 in 10 were recorded.

Other members of the Solanaceae which were successfully infected by grafting were: -- Eggplant var. Black Beauty: on this host the virus produced chlorosis and stunting with necrotic flecks on some plants; Sweet pepper var. Fordhook: here the symptoms consisted of dark green leaf mottle with yellowing along the veins and with interveinal chlorotic patches developing later; Tobacco var. White Burley: this host showed a leaf mottle with yellow chlorotic patches between the veins in older plants; Nicotiana glutinosa: this showed a variegated light and dark leaf mottling with some puckering of young leaves.

White fly (Aleyrodidae) is a common pest in Trinidad greenhouses and it was noticed during the present investigation that plants grown from seed in insect proof cages and kept entirely free from white fly did not become infected. White fly is therefore to be suspected as a vector of the virus of tomato bronze leaf in Trinidad. It may be noted here that this disease closely resembles

[1]Work reported in this paper was conducted by the writer in 1949.

Table 1. Symptoms on selected hosts caused by the viruses of four diseases of Solanaceous plants in Trinidad, B. W. I.

The Virus Diseases	Tomato	Eggplant	Pepper	Nicotiana glutinosa	Nictoiana tabacum
Tomato twisted leaf	Growth checked. Downward twisting of leaves. Veinal necrosis in var. Ponderosa. Growth resumed in a few days but leaves remained small and showed interveinal chlorosis.	Not susceptible	Capsicum annuum caused mottling of leaf with blistering of leaf	Not susceptible	Not susceptible
Tomato bronze leaf	Bronzing of young leaves. Necrosis and die back of growing points.	Var. Black Beauty. Chlorosis and stunting with necrotic flecks in some cases.	Var. Fordhook. Dark leaf mottle, interveinal chlorotic patches.	Light and dark leaf marking with puckering of young leaves.	Var. White Burley. Leaf mottle with interveinal chlorotic patches in old plants.
Eggplant mosaic	Growth check. Leaf crinkle and mottling.	Yellow lesions. Vein clearing of expanding leaflets. Irregular light and dark green mottling.	Sparse irregular chlorotic lesions.	Circular necrotic lesions followed by chlorotic ring spots on young leaves. Stunting of plant, especially seedlings. Mottle, wavy leaf margins and vein banding in some cases.	Not susceptible
Sweet pepper mosaic	Not susceptible	Not susceptible	Vein clearing followed by leaf mottling. Later leaves showed vein banding and leaf mottle. Laminae small and crinkled. On var. Bell Hot growth stopped; necrosis and death of plant ensued.	Not susceptible	Var. White Burley. vein clearing and banding. Slight mottle and crinkle of leaf.

the widespread tomato spotted wilt in its symptoms. Spotted wilt is known to be spread by thrips, but no indication that thrips act as vectors of the virus of bronze leaf of tomatoes has been observed in Trinidad. However, on experimental plants and on zinnia in private gardens, symptoms suggestive of spotted wilt are often seen. All attempts to transmit the symptoms of spotted wilt to tomato plants have proved unsuccessful.

3. EGGPLANT MOSAIC: In Trinidad this common virus disease of eggplant produces irregular light and dark green leaf mottling but little damage results to the imported Black Beauty variety, or to the local hybrids. The virus is readily sap-transmissible and it was quite a normal occurrence to obtain 100 percent positive results by rubbing the leaves of test plants with infective sap, using carborundum powder as an abrasive. Chlorotic yellow lesions appear on the leaf in about three days and a distinct vein clearing of the expanding leaflets develops six to ten days after inoculation. The characteristic irregular mottling then appears. It has been demonstrated by Dale (personal communication) that a flea bettle (Epitrix sp.) is a vector of the disease.

Other plants were experimentally inoculated by rubbing their leaves with the infective sap. On tomato symptoms appeared in six to eight days. These consisted of a temporary check to growth, which was soon followed by crinkling and the appearance of light and dark green mottling of the leaves. The symptoms tended to become less obvious as the period of infection increased. The red currant tomato (Lycopersicon pimpinellifolium) gave similar symptoms when inoculated with the virus. Inoculations on to the leaves of the potato (Solanum tuberosum) var. Arran Pilot produced sparse local lesions on the laminae, which later became necrotic. The next leaves to expand showed similar but more numerous chlorotic spots which also became necrotic. Subsequent leaves showed a light and dark green leaf mottling but no lesions. Inoculated plants of sweet pepper developed sparse irregular chlorotic lesions. These were up to 8 mm. in diameter and had indefinite margins. Similar lesions appeared on the next leaves to expand, but subsequent leaves were symptomless, although the virus when recovered from them proved infective again to eggplant. Nicotiana glutinosa, when inoculated, gave circular necrotic lesions in two to four days, followed by chlorotic ring spots on leaves not fully grown at the time of inoculation. The next leaves to expand showed vein clearing, and on subsequently produced leaves a dark and light green mottle appeared. The dark areas which sometimes bordered the main veins gave the effect of an irregular vein banding. On some infected plants the leaf margins were wavy, and in seedlings especially, a general stunting of the young plants was observed.

The following species were tested and found not to be susceptible to the virus of eggplant mosaic: Tobacco, Nicotiana rustica, N. sylvestris, N. sanderae, N. alata var. grandiflora, N. chinensis; cucumber (Cucumis sativus); pumpkin (Cucurbita pepo).

4. PEPPER MOSAIC: A disease of peppers in Trinidad caused by the tobacco mosaic virus was described by Dale in 1943 (1). The most striking symptoms of this infection were a chlorosis and necrosis of the leaves accompanied by abscission not only of the leaves and flowers, but also of any young fruits which may have developed.

It is noteworthy that infection of peppers by the above virus has not been observed in the field during recent years. However, another virus disease of peppers, the symptoms of which are distinct from those produced by the tobacco mosaic virus, is now of common occurrence in Trinidad. On experimental plants the first symptoms, which appear 8 to 12 days after inoculation, consist of a vein clearing of the expanding foliage followed by a mottling of the leaves. On later formed leaves a striking feature is the appearance of an irregular and discontinuous vein banding, with a dark and light green mottling of the interveinal areas. Moreover, the laminae are reduced in size and present a somewhat crinkled appearance. Fruits of infected plants also tend to be smaller than normal and are often abnormally twisted and blistered, this being more marked in the mild, large fruited sweet peppers (Capsicum annuum var. grossum) than in the hot peppers (var. minimum). Perennial "bird peppers" and "cherry peppers" (C. frutescens), which are common enough in Trinidad gardens, also frequently show leaf mottling and crinkle symptoms similar to those described above on C. annuum. Cross inoculation experiments on these various hosts showed that the same virus is responsible for the disease, and it is not unlikely that these infected perennial plants play a part in the spread of the mosaic to new plants of annual peppers. The "Large Bell Hot" variety of hot pepper, when infected with the virus of the Trinidad pepper mosaic, developed striking and characteristic symptoms after seven days; growth ceased early, and with rapid necrosis and defoliation of the plant setting in, death of the entire plant soon followed. These symptoms bear a striking resemblance to those reported on this variety of pepper from Puerto Rico (2).

Inoculations with the virus of the present pepper mosaic disease were also carried out on White Burley tobacco (Nicotiana tabacum), by rubbing with sap from infected peppers. Symptoms appeared in ten days on about 60 percent of the plants, the virus apparently being less infective to tobacco than to pepper plants when transmitted by rubbing. First symptoms on tobacco consisted of a vein clearing followed by an irregular dark green vein banding of the leaves. Mottling of the foliage here was less marked than that produced by the virus on peppers, and any leaf crinkle, if it appeared at all, did not develop until a later stage and was moreover much less marked. Similar symptoms were produced by the same virus on an ornamental tobacco variety Nicotiana tabacum var. macrophylla, and on N. chinensis. Species which proved to be immune to infection with the virus of pepper mosaic were Lycopersicon esculentum, Solanum melongena, Vigna unguiculata, Nicotiana glutinosa, N. rustica, and N. sylvestris.

COMPARISON OF VIRUS PROPERTIES

1. Table of Physical Properties

	Heat inactivation (° C).	Dilution tolerance	Longevity in vitro
Virus of eggplant mosaic	65	1:10,000	3 weeks at 15°C.
Virus of tomato twisted leaf		Not sap-transmissible	
Virus of tomato bronze leaf	40	Over 1:2,000	85 minutes at room temp.
Virus of pepper mosaic	62	1:5,000	16 days (15° C.)

2. Cross Immunity Tests

A complete series of cross immunity tests were carried out on the viruses of tomato twisted leaf, tomato bronze leaf, eggplant mosaic, and pepper mosaic. None of these viruses conferred immunity to subsequent infection by any of the others.

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DISEASES IN CROFT LILY PLANTINGS, 1949-50[1]

Frank P. McWhorter[2] and C. J. Anderson[3]

This report is based on the regulatory field inspections made in Oregon by the Oregon Nursery Service and on field and laboratory studies made by McWhorter.

The principal diseases of Lilium longiflorum Thunb. continue to be scale tip rot, botrytis blight, scorch, and dieback. Of minor importance are the streak-fleck complex and the Pacific Northwest form of Colletotrichum scale rot. Meadow nematodes (Pratylenchus spp.) a comparatively new menace to the industry, are not present in four Oregon locations. The presence of meadow nematodes in roots of Croft lily was noted in 1946 in a California planting (1).

Scale Tip Rot

Although there is apparently less scale tip rot than formerly, this rot is still present to some extent in every field near the coast. During 1949 an attempt was made to correlate the degree of rot with fertilizer variants. All the bulbs from a complete fertilizer and minor element test were sorted and graded with respect to degree of rot. This involved the grading of about 6,000 bulbs, grown as 54 lots, representing 14 treatments. No treatment showed any significant increase or decrease in the amount of this rot. This was disappointing since there has been some evidence that boron is a limiting factor for scale tip rot. There was no correlation of the amount of rot with boron even in the special lots that received 150 pounds of borax to the acre.

It is suggested that the fact that there is less scale tip rot than formerly correlates with the great increase in irrigation of lily plantings during the last three years. That scale tip rot is largely a "drought spot effect" remains the best explanation of the disease.

Botrytis Blight and Scorch

A few fields were severely injured by botrytis blight this season. There was poor correlation between the absence of botrytis blight and the application of bordeaux spray. Many of the fields that were free from botrytis blight received no spray.

The determination of botrytis blight damage is now complicated by our recognition of the physiological disease called scorch, which frequently affects Croft lilies in greenhouses (3). It is suggested that the probable presence of scorch in field plantings explains those cases where severe blight occurs in fields well protected by bordeaux spray. It is probable that a considerable portion of the injury formerly attributed to botrytis blight may have been due to the physiological disease, scorch.

Dieback and Bunchy Top

During the early 1940's when the growing of Croft lilies became a major industry, dieback and bunchy top were abundant and serious. Both occurred separately on different plants and together on the same plants. Following the general practice of using hot water treatment, the bunchy top form has disappeared and only occasional outbreaks of the dieback form occur. The recommendation that bulblets be treated every season has given excellent economic control. The survival of the causal nematode (Aphelenchoides olesistus (Ritzema Bos)) in the soil is so low that the usual crop rotation suffices and special soil treatments have never proved necessary in Oregon and California plantings.

Streak-Fleck Complex

A three-year study of the streak-fleck complex occasionally found in Croft lily plantings has been completed at the Oregon Agricultural Experiment Station. The many forms of this disease can be explained as caused by variants of western cucumber mosaic virus. Although the virus is widely distributed, occurrences of the disease in Croft plantings are rare. The disease was found in six fields during 1950. None of these were in the chief lily areas south of Bandon, Oregon; all were in northern Oregon. The distribution pattern closely follows that of commercial cucumber plantings. The source of the causal virus, however, is not cucumbers but the same overwintering host plants that are primary sources of infection in cucumber fields. The principal overwintering hosts of Marmor cucumeris H., which serve as reservoirs of the virus in the

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[2]Plant pathologist, Oregon Agricultural Experiment Station, and Agent, U. S. Bureau of Plant Industry, Soils and Agricultural Engineering, Beltsville, Maryland.

[3]Plant Pathologist, Oregon State Department of Agriculture.

eastern United States, are not present in the Pacific Northwest. In several instances Primula spp. have been the probable sources of infection of lily plantings in Oregon and California.

Colletotrichum Scale Rot

A very mild form of Colletotrichum scale rot was found in most fields during 1950. The bulbs are not so severely injured as are the Creoles by a similar Colletotrichum in Louisiana (2). They are seldom injured to a degree that is noticed by the growers. A study of the relation between the eastern and western strains of Colletotrichum is being made by Dr. A. G. Plakidas at Louisiana State University.

Meadow Nematodes

The presence of meadow nematodes (Pratylenchus spp.) in lily roots was first discussed by Butterfield (1). The occurrence of these nematodes at three Oregon commercial plantings was

FIGURE 1. The general effect of the meadow nematode on the growth of lilies in once good lily land. The same area in 1947 and 1950.

confirmed by Mr. W. D. Courtney and the very serious infection in the experimental plots near Port Orford was confirmed by Mr. Gerald Thorne.

The symptoms of meadow nematode infection have not been adequately determined. A study of the pathology of these nematodes on the Croft lily will be made by Dr. H. J. Jensen at the Oregon Agricultural Experiment Station. Infestation by Agasphaerops nigra Horn, by symphilids, by Aphelenchoides olesistus, and by meadow nematodes all may lead to the presence of dead plants that superficially appear to have died from a common cause. Also, the killing of young plants by Botrytis is often confused with all of these. Hence the need for an adequate record of the progress of the symptoms caused by meadow nematodes that recently have invaded lily plantings.

An estimate of the potential importance of meadow nematodes in lily cultures can be made from the following case history. A field of grower "X" produced a superb lily crop in 1947 (see Fig. 1). No disease of any kind was present. The same field was replanted in the fall of 1948 for the 1949 crop. This 1949 crop failed commercially. The field was sprayed approximately ten times with bordeaux during 1949, but the plants continued to blight with symptoms that resembled botrytis blight. No signs of botrytis blight were observed in leaf samples from this 1949 field when typically blighted leaves were examined by laboratory methods. It was therefore concluded that the plants in this field were injured by the physiological disease, scorch. Therefore this field was chosen (fall 1949) as a preferred location for a comprehensive trial of fertilizer applications to prevent scorch. During October 1949, a fertilizer test involving thirteen variables was planted in this field with 3,000 bulbs from a distant field that evidenced no disease. The planting stock was excellent and had an expected bloom of three flowers per plant. In the early spring of 1950 there was an even stand of normal Croft lily plants. By late April, however, yellowing and dying of foliage were apparent and the plants became progressively worse so that by June the foliage of many plants was dead. None of the plants attained more than one-third of their expected size, only a few bloomed and most of the plants appeared entirely dead by early September. These foliage symptoms were accompanied by rotting of the stem rootlets. Samples of these plants sent to Mr. Gerald Thorne were reported infected with Pratylenchus sp. with the comment that hundreds of nematodes were present in the sample sent him. The fertilizer experiment was a total failure.

The origin of this nematode invasion is an exciting challenge for investigation. The fertilizer plot was located high on a mountain slope far above any other cultivated area and several miles from other lily fields. Above the field was cut-over forest land that has never been cultivated. It is probable that the nematodes are entering lily plantings from some unknown native host.

At this time, these nematodes are known to be present in one California and four Oregon locations. Since they are potentially the most destructive pest that has appeared in these lilies, a better understanding of their symptomology is imperative for making adequate protective surveys.

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CONTRIBUTION OF THE DEPARTMENT OF BOTANY AND PLANT PATHOLOGY, OREGON AGRICULTURAL EXPERIMENT STATION, AND THE BUREAU OF PLANT INDUSTRY, SOILS AND AGRICULTURAL ENGINEERING, UNITED STATES DEPARTMENT OF AGRICULTURE, BELTSVILLE, MARYLAND

GLADIOLUS DRY ROT TESTS IN WESTERN WASHINGTON

Charles J. Gould[1]

Dry Rot and Botrytis Rot have long been our most important corm diseases of gladiolus in western Washington. With the development of proper curing techniques and other practices, Botrytis Rot can be eliminated as a major menace. Dry Rot, however, continues to be a serious threat. Corm treatment with Tersan has been found to give consistently good results, but not quite as good as desired. Likewise its cost is high. Consequently the search for a cheaper and better treatment has continued.

In our 1950 tests in western Washington, a stock of Picardy was used that was very severely infested with Dry Rot. All obviously diseased corms (No. 5) were discarded and the others were dipped for one hour before being planted on May 9. The soil was a sandy loam, well-drained, with water supplied from overhead irrigation.

The Tersan- and Natriphene-treated corms appeared stimulated by the treatment, whereas the Mersolite W treated corms were definitely retarded. This was evident both in rate of emergence of plants from the ground and in rate of blooming (Table 1). Mersolite is a mercury compound (phenyl mercuric acetate) and its retarding effect was therefore not surprising since we had often seen it happen with other types of mercury treatments of corms in western Washington.

Table 1. Results of dry rot tests in 1950 in Western Washington.

Material	Concentration 1 lb. in:	No. spikes cut by Sept. 15	No. spikes cut by Oct. 20	Av. height of spikes inches	Wt. in lbs. of healthy corms	Av. wt. per healthy corms in lbs.	No. of large (No. 1) corms	Percent of healthy corms by wt.
None	--	152	257	35.8	9.4	.057	56	33.3
Tersan	6 gal.	241	427	36.9	26.8	.063	188	71.3
Natriphene	96 gal.	204	385	35.8	14.3	.059	77	44.4
"	193 gal.	185	370	35.1	11.8	.063	67	37.7
Mersolite W	241 gal.	31	114	39.7	29.0	.050	90	97.3
"	482 gal.	85	221	39.7	31.7	.054	145	94.3
"	723 gal.	95	260	39.8	33.5	.056	124	93.1

What was surprising, however, was the condition of the corms at harvest time. The Mersolite plots produced the largest yields of healthy corms, followed by Tersan, with the Natriphene being almost as poor as the untreated sets. Although there were more healthy corms in the Mersolite sets, the number of large corms was greatest in the Tersan set.

The total yield of healthy corms and their average weight increased as the concentration of Mersolite was reduced even though the percent of diseased corms also increased slightly.

The Mersolite definitely appears to be promising for Dry Rot control, although additional tests will be made before recommending it for general use in western Washington. The delay of flowering is particularly important to our cut-flower growers. The above treatments were intentionally made an hour long in order to intensify the control and aggravate any possible injury. Perhaps the delay in flowering can be overcome without decreasing disease control by reducing the length of time of treatment to five or ten minutes, by using the Mersolite at a more dilute concentration, or even by mixing some Tersan (which seems to stimulate) with the Mersolite. These possibilities will be tested during the coming season. Meanwhile we are continuing to recommend the use of Tersan (1 lb. in 6 gals. of water for 10 minutes) or its dry form Arasan (as a dust) for Dry Rot control in western Washington.

WESTERN WASHINGTON EXPERIMENT STATION, PUYALLUP, WASHINGTON

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SPHACELOMA SCAB, A NEW DISEASE OF SOYBEANS DISCOVERED BY PLANT PATHOLOGISTS IN JAPAN

Anna E. Jenkins

A list of the "major food crop diseases" in Japan published in 1949 (6, p. 424) includes under "Vegetables" one disease of soybean (Glycine max (L.) Merr.), namely anthracnose, caused by Colletotrichum glycinis Hori. One of the five diseases listed under "Fruit" is scab of citrus fruits caused by Elsinoë fawcetti Bitanc. & Jenkins.

A disease of soybean corresponding to citrus scab has now been reported in Japan as I have been advised through recent interchanges of correspondence relative to "spot anthracnoses", [1] with T. Togashi, Professor of Plant Pathology, Agricultural Institute, Yokohama National University, Yokohama, Japan.

In order that prompt mention of the soybean disease may be made in the Plant Disease Reporter, Professor Togashi has graciously furnished me with an English translation of Kurata and Kuribayashi's abstract of their preliminary paper on the subject (4). Following the translation, Kurata's (3) article based on data from the same source as the abstract, was cited, together with Endo's (2) report of field observations on the disease. The following account of the disease is taken from the translation, and the accompanying illustrations (Fig. 1) are from specimens contributed by Professor Togashi.

In 1947 Kuribayashi discovered a destructive soybean disease which was hitherto unknown and which has been spreading locally and damaging the soybean crop in the Nagano Prefecture. In the field, leaves, stems and pods are affected. In severe cases of pod infection the seed may fail to develop in varying degrees. The disease was named "Kokuto-byo" (scab disease) by Kuribayashi.

Lesions on leaves, stems, and pods are hyperplastic in nature. On leaves they are circular to irregular, frequently visible on both leaf surfaces, usually more or less raised above, commonly "vinaceous buff", [2] often fading to "pale drab gray", minute to about 4 mm. in diameter. Spots on stems range from minute to elliptical-elongate areas generally "vinaceous buff", sometimes with reddish brown margins, reaching at least 1 cm. in length or 2 cm. when confluent. On pods young lesions are generally red to "Kaiser brown", upon maturity becoming "dark olive" to black, with paler centers and reddish brown margins.

In response to the writer's specific inquiry, Professor Togashi wrote that the symptoms of the scab on soybean pods are comparable to those, for example, of scab of lima bean (Phaseolus limensis Macf.) [3] caused by Elsinoë phaseoli Jenkins [4].

Kurata and Kuribayashi reported that Sphaceloma sp. was isolated from leaf, stem and pod lesions of soybean and that inoculations on soybean with the organism gave positive results.

Not only is this new Sphaceloma host "a cultigen of great antiquity" in the East (cf. Ames 1, p. 55), but it also ranks high among "major agricultural crops of the United States " (cf. McCallan 5, Table 1).

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4. _____ and K. Kuribayashi. New scab disease of soybeans, caused by a species of Sphaceloma. Paper read at the Annual Meeting of the Phytopathological Society, Japan, held in Tokyo, April 7, 1950. Ann. Phytopath. Soc. Japan 15, No. 2, In press. Abstract (In Japanese).

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[1] General term for plant diseases caused by Elsinoë, of which the conidial stage is Sphaceloma (cf. Jenkins, Plant Dis. Reptr. 31: 71. 1947). For revised descriptions of these two genera cf. Jenkins and Bitancourt (Mycologia 33: 338-340. 1941).

[2] This color reading and others here placed within quotation marks doubtless are based on Robert Ridgway's Color standards and color nomenclature (43 p. Washington, D. C. 1912).

[3] cf. Jenkins, Jour. Agr. Res. 42: 13-23. 1931.

[4] Described in Jour. Agr. Res. 47: 788. 1933.

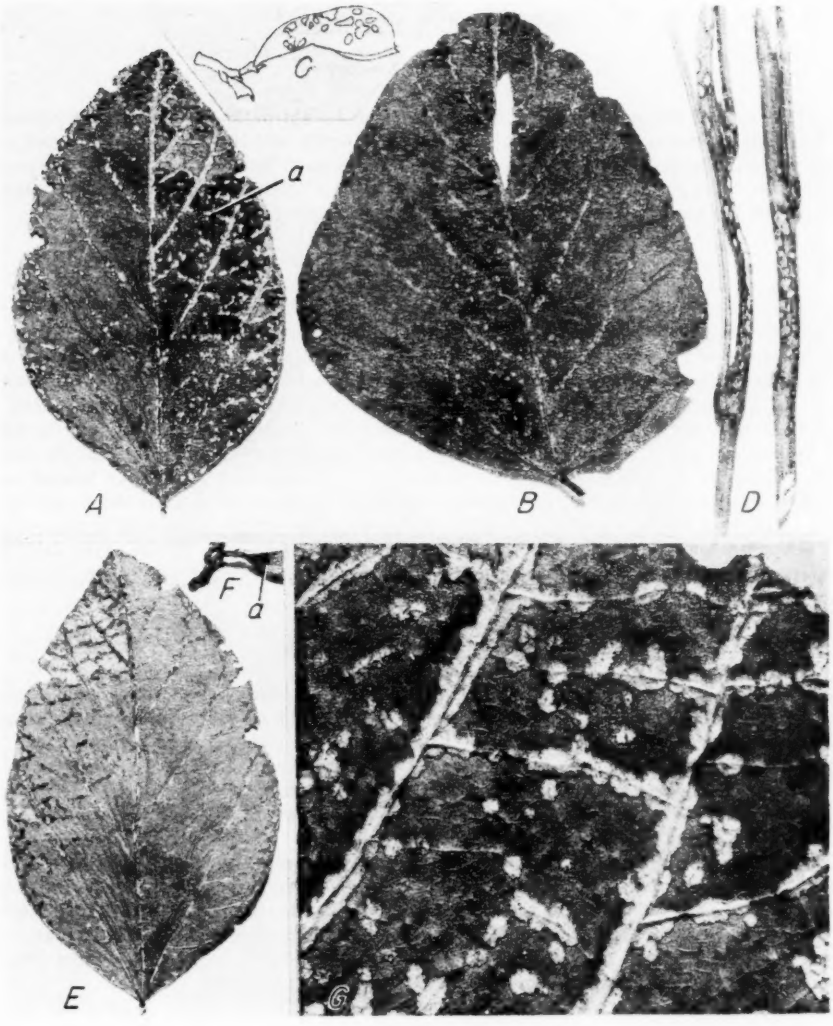

FIGURE 1. Scab on soybean leaves (A and B), pod (C), and stems (D) grown in Japan; X 3/4. E, reverse of A; X 3/4. F, a, lesion on other valve of pod shown in C; X 3/4. G, same as A, a; X 3 3/4. A and B, from Naniai-mura, Naganoken Prefecture, Aug. 8, 1950, H. Kurata. C, from Imoi-mura, Nagano Prefecture Oct. 16, 1950, K. Kuribayashi. Photographs by R. L. Taylor.

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DIVISION OF MYCOLOGY AND DISEASE SURVEY, BUREAU OF PLANT INDUSTRY, SOILS, AND AGRICULTURAL ENGINEERING

SURVEYS FOR OAK WILT [1]

Marvin E. Fowler

Oak wilt is one of the most important problems in forest pathology today and presents a serious challenge to those of us who are interested in trees and their health. A brief review of our present knowledge about oak wilt reveals that it has been known as a definite disease for a number of years. Its pathogenicity was proved by inoculation experiments in Wisconsin in 1942. Henry described the causal fungus and named it Chalara quercina.

Symptoms of the Disease

Oak wilt produces very noticeable leaf symptoms, which vary with the oak species affected. Early symptoms may be limited to a wilting and bronzing of foliage only in the upper crown, usually followed by wilting and bronzing of the foliage throughout the crown, beginning at the top and ends of lateral branches and accompanied by partial defoliation. In a more advanced stage the majority of the leaves are dead and defoliation is more pronounced. In some cases very little defoliation may occur when a tree dies very rapidly. Some leaves may stay on the tree to the end of the season and a few may even persist until the following summer. In the advanced stages of the disease, severe defoliation is common. The bronzing of red oak leaves usually begins at the apex and lobes of the leaf blade, spreading to the midrib until the entire leaf blade is involved. The leaf surface near the midrib frequently has a water-soaked appearance before bronzing. Affected leaves in any stage, including those that are fully green, may fall from the tree. Sprouting along the trunk and larger branches frequently occurs before an infected tree dies. Small trees do not escape from oak wilt. (Fig. 1, A, B).

Susceptibility of Oak Species

We know of no native oak species that is immune to this disease. Although the disease has not yet been found in nature on all of our oak species, inoculation tests on a number of oak species have indicated that they are susceptible and none are yet known to be immune. In nature the disease kills red and black oaks rapidly and white oaks more slowly. On white oaks the disease usually affects only individual branches in any one season, rather than the entire crown as in red oaks. The following year additional white oak branches may be affected and the entire tree may die within three years.

Inoculated trees of the black oak group displayed symptoms within 15 to 30 days and the trees died about 60 to 90 days later. Short-lived sucker growth frequently appeared on the trunks and branches of trees of this group in the spring following late season inoculation. In the white oak group, usually infections are confined to the inoculated branches during the first year. The disease may persist and the tree may die within a few years. Some research workers isolated the fungus from stumps two years after the trees were killed by oak wilt.

New Hosts

In 1950, willow oak (Quercus phellos) and southern red oak (Q. rubra) were found for the first time to be naturally infected. It is also interesting to note that the oak wilt fungus was isolated from diseased Chinese chestnut trees (Castanea mollissima) growing in a plantation in Missouri.

History of the Disease

The history of oak wilt is somewhat obscure. An examination of records made and reports of dying oaks in the Lake States force one to concede that oak wilt probably has been present in Wisconsin for at least 20 years or more, but we cannot establish this with certainty. We know that oaks were dying in Wisconsin and that a competent forester described symptoms in 1929 that are not unlike those we recognize as oak wilt today. Oaks may have been dying in the same manner many years earlier.

Until the summer of 1949 oak wilt was known only in Wisconsin, Minnesota, Iowa, Illinois, and Missouri. In 1949 it was found further south in Missouri, at the northern edge of the Ozark region, and also in northwestern Indiana. It was the finding of oak wilt in southern Missouri that

[1] Based on an address given at the Silver Anniversary Meeting of the New Jersey Federation of Shade tree Commissions at Atlantic City, New Jersey, January 22, 1951.

FIGURE 1. A, Black oak with a 10-inch d.b.h., which died following inoculation. B, A group of oak trees that died in nature, with symptoms of oak wilt. C, An infected black oak twig with bark removed, showing sapwood streaking that is sometimes present. D, Natural root grafts of white oak, through which the disease can spread from a diseased to a nearby healthy tree.

Illustrations from Phytopathology 34: 636-647. July 1944.

caused great alarm. Heretofore the disease was known only in the more northern part of the oak range where oaks had been dying from various causes for years, attributed often to adverse climatic conditions. But in southern Missouri the disease was already in the northern part of a vast oak forest region where the climate is believed to be conducive to oak growth. This oak forest is of great economic importance, extensive in area, running south through the Ozarks and down into the delta bottomlands almost to the Gulf of Mexico. The forests of the Ozarks are predominantly oak and their destruction by disease would constitute a major catastrophe to forestry in that vast region. Furthermore, the presence of oak wilt so far south demonstrated that the disease was not climatically limited to cooler areas as had been hoped previously.

The discovery of oak wilt in southern Missouri in 1949 led to speculation regarding the spread of this dread disease. Had the disease suddenly jumped southward to the northern edge of the vast Ozark Mountain oak forests? Would it spread rapidly through these valuable oak stands? The need for a comprehensive survey to determine the extent and intensity of the disease was apparent. Information on the prevalence and distribution of the disease was essential before the seriousness of the problem could be evaluated. An allotment of funds made available by the Forest Pest Control Act enabled the Division of Forest Pathology to initiate a limited survey for oak wilt in July 1950.

A comprehensive survey could not be made because of the limited time and personnel available and the large area to be covered. The objectives were to determine how far south the disease occurred, how long it had been present in the area, and the prevalence of oak wilt near its southern limits. It was recognized from the beginning that surveys made from automobiles or on foot would be inadequate and slow. Only a small fraction of the oaks can be seen from roadways and a survey made in this manner would at the best be only a spot survey and would permit numerous infections to remain undiscovered.

Airplane Survey

Because of the pronounced leaf symptoms of oak wilt it was thought that scouting for the disease from airplanes might be feasible. The Division of Forest Pathology rented a commercial Stinson airplane in July 1950 and made flights over a known oak wilt infection in southeastern Missouri. Healthy and diseased oaks were readily distinguished in the forest stand, even at considerable distance. Therefore we proceeded to use airplanes of various types during the remainder of the 1950 season to survey for oak wilt in southern Missouri, northern Arkansas and in western Kentucky and Tennessee. Most of the flights were made at 100 to 200 feet above the tree tops. No attempt was made to survey the entire area. Instead, flights were made east and west across southern Missouri and northern Arkansas in strips about 50 miles apart (Fig. 2). Similar flights were made approximately north and south through the extreme western parts of Kentucky and Tennessee and down into eastern Arkansas. The observations were more or less limited to areas readily accessible from passable roads. This enabled the ground crews to examine more easily the suspicious trees. Nevertheless, most of the trees spotted by the aerial observer would not have been visible from an automobile on the nearest road. A more intensive aerial and ground survey will be necessary to determine the intensity of the disease in the area.

The 1950 surveys were made in Missouri in cooperation with the Missouri Agricultural Experiment Station and in Arkansas with the Division of Forestry and Parks of the Arkansas Resources and Development Commission. The Bureau of Entomology and Plant Quarantine of the U. S. Department of Agriculture rendered valuable assistance to the survey by providing a Cessna 195 airplane with pilot and an observer for about ten days.

In an airplane 200 to 500 feet above the tree tops the observer has a better view of the tree crown than is usually possible on the ground unless he is directly beneath the tree. The bronzed foliage of diseased trees stands out vividly in contrast with the green foliage of the surrounding healthy trees. A surprising feature of scouting for oak wilt from a low flying airplane is the ease with which one can see down through the dominant tree crowns to the foliage of the suppressed trees beneath. The understory trees can be seen best when flying from 100 to 200 feet above the tree tops. Oak wilt foliage symptoms are most conspicuous from mid-June to early September. Some trees may develop symptoms earlier and most can be distinguished in the fall until the normal autumn foliage colors begin to develop.

While it is easy to distinguish discolored from healthy green foliage from a plane, it is not always easy to distinguish between oak-wilted trees and trees dying from other causes. There are a number of causes of wilting, browning, and dying of oak leaves. Perhaps the most confusing symptoms are those induced by lightning strikes and by the poisoning and girdling of unwanted trees. In both these instances the trees may die suddenly while in full leaf. The leaves on such trees brown over the entire surface, and very little or no defoliation takes place until near the

FIGURE 2. Oak wilt aerial survey, 1950

end of the season. By contrast, oak-wilted trees are usually not as brown due to parts of leaf blades remaining green. Also oak-wilted trees may undergo partial defoliation and the crowns appear thin by comparison. A careful observer soon feels that he is able to recognize oak wilt with reasonable accuracy. However, to be certain that oak wilt is present in a new location, it is necessary to culture the Chalara fungus from material collected from the suspected tree.

Green or recently dead wood that has not completely dried out is satisfactory for culturing. Twig or branch specimens, 6 to 8 inches long and 1/2 to 1 inch in diameter, should be collected from about six places on the tree. Sapwood streaking is often present in infected trees, but some infected branches show no discoloration. The oak wilt fungus can sometimes be isolated from clear wood as well as from wood with discolored streaks. (Fig. 1, C).

Airplane scouting for oak wilt enabled a few men to inspect a much larger forested area in a few weeks than would have been possible in many months on the ground. Areas that did not contain wilted trees were eliminated from further consideration, and ground crews were sent into the forests only where wilted or dying trees had been observed from the air. It is often necessary to make an almost 100 percent cruise of a dense forest to find scattered wilted trees that could be seen almost at a glance from a low-flying airplane. When a suspicious tree or group of trees is seen from the air, the observer marks the location as accurately as possible on a map and relates it to landmarks that can be recognized by a ground crew. In conducting a survey to determine if the disease is present in an area, the ground crew needs to visit only selected suspicious trees to collect specimens for culturing.

Relatively low-speed (80-plus miles per hour air speed) aircraft such as Piper, Stinson, Aeronca, Luscombe, and Cessna types are satisfactory for survey work. The plane must be so constructed that the observer will have a good view of the forests. Helicopters would be very

satisfactory also, and this type of airplane has been successfully used on the Cook County Forestry Preserve in Illinois, but the hourly rental for this type of plane is higher than for others and its slow speed is not deemed essential to some types of scouting.

The most satisfactory airplane used in our 1950 survey was a Government-owned and operated Cessna 195. This airplane had been modified by the installation of plexiglass doors on each side, one extending to the floor of the cabin and the other to within one foot of the floor. This modification afforded excellent lateral visibility. The smaller door could be opened in flight for taking oblique photographs and there was a camera port in the floor of the cabin for taking vertical photographs. This plane was powered with a 300 horse power engine and had a variable pitch propellor which enabled it to climb at a very much faster rate than the other planes used. The rate of climb is important for getting off of small airstrips, out of narrow valleys, and over steep ridges, powerlines, radio towers, tall trees, etc.

Distribution of the Disease

The survey conducted in 1950 revealed that oak wilt is rather generally scattered and well established in southeastern Missouri. It appears to be less prevalent west of a line projected

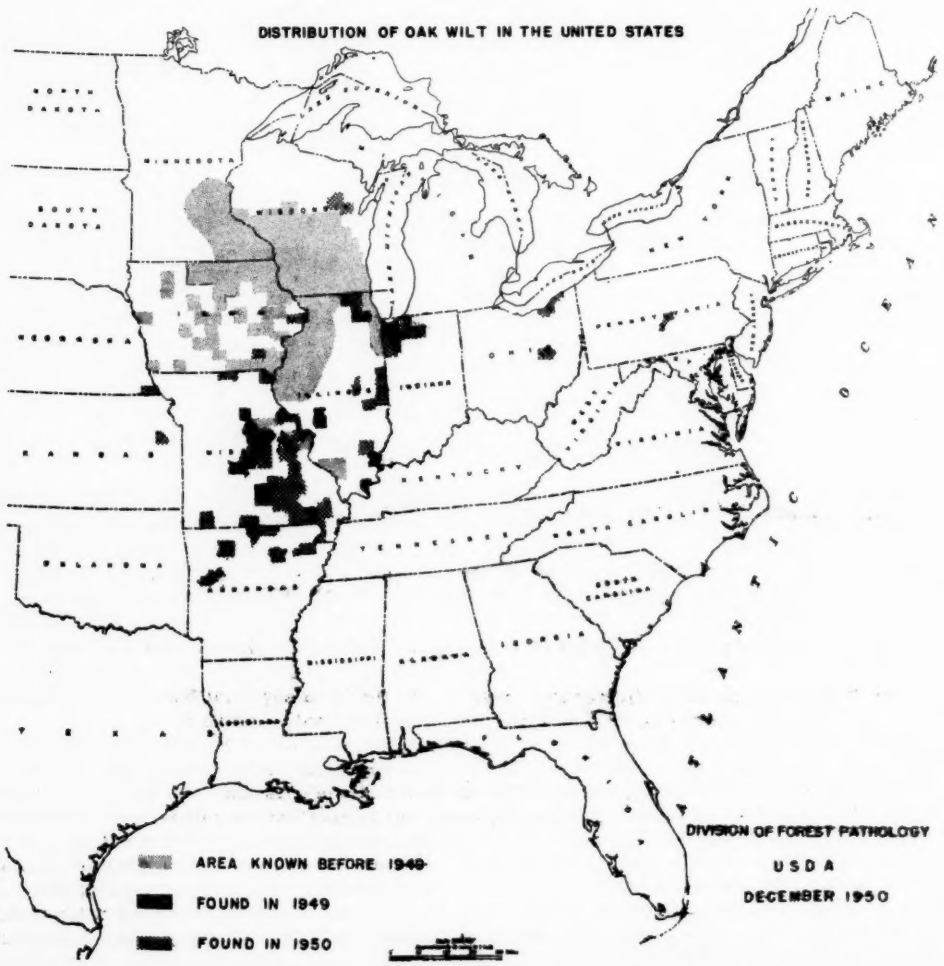
DISTRIBUTION OF OAK WILT IN THE UNITED STATES
AREA KNOWN BEFORE 1949
FOUND IN 1949
FOUND IN 1950
DIVISION OF FOREST PATHOLOGY
U S D A
DECEMBER 1950

FIGURE 3. Distribution of oak wilt in the United States, December 1950.

from the Lake of the Ozarks southward to the Winona District of the Clark National Forest. The disease was spreading actively in southern Missouri in 1950. Numerous individual recently diseased trees were found in otherwise healthy stands. At one location near Poplar Bluff, Missouri, an infected area was found in 1949. A 160-acre tract around this infection center was cruised in 1950 and nine separate and distinct infection centers were found. These nine infection centers were readily visible when a flight was later made over the area. The condition of the dead trees in the largest infection center in this tract indicated that the wilt had been present for about five years. Additional evidence to support this conclusion was obtained from an examination of trees released by the death of the oaks. The released dogwood and hickory trees showed marked increase in width of annual rings beginning with 1945.

During 1950 oak wilt was definitely established in 19 additional counties in Missouri. About 80 percent of these were found by the survey crew of the Division of Forest Pathology. This crew also found the disease in six counties in Arkansas.

An examination of infections revealed that the disease has been present in southern Missouri for possibly five years or more, but no large or old infection centers were found in Arkansas. No infections were found in Arkansas that appeared to be more than three years old. Further evidence may show that the disease has recently entered that State and that it is spreading southward. The southernmost confirmed cases of oak wilt were found near Boonville, in Logan County, Arkansas (Fig. 3).

Other investigators found oak wilt in additional new sections of the country during 1950. The most significant discoveries that extend the known range of the disease were in Richardson County, Nebraska, in Lake, Cuyahoga and Coshocton Counties, Ohio, and in Mifflin County, Pennsylvania. Oak wilt was also recently found in Douglas County, Kansas, as reported on page 119 in this issue of the Reporter.

Control of the Disease

The means by which the oak wilt fungus spreads from one location to another to establish a new infection center is not known. No one has yet found the fungus fruiting externally on dead or dying oaks. The pattern of spread does not suggest wind-disseminated spores, but it is not known if it is spread by this means or by some insect, bird, rodent, or other means. Until we understand how this is accomplished we have no basis on which to recommend measures for control of this type of spread. Sanitation through destruction by burning of diseased trees has been suggested.

Riker and his associates in Wisconsin found that the local spread of oak wilt that results in an enlargement of an infection center is accomplished by the transmission of the fungus underground, through the natural root grafts that commonly occur between oaks of the same species growing close together (Fig. 1, D). Thus the disease may spread in a more or less circular pattern from the diseased trees to a few or a considerable number of the surrounding healthy trees. In five or ten years a pocket of dead trees from 1/8 to 1 acre in extent commonly develops.

The spread of the disease through root grafts may be stopped by poisoning the healthy trees around the perimeter of the diseased trees. Sodium arsenite and Ammate have been used. Great care must be exercised when using sodium arsenite because the chemical is attractive and poisonous to animals. Ammate does not kill trees as rapidly as the arsenite, but is safer to use.

Chemicals sometimes not only kill the poisoned trees but also others that are connected to the treated trees by root grafts. This control method may be undesirable on some street, lawn, or park trees. Mechanical severance of roots connecting diseased and healthy trees has also been successful in preventing spread of the disease. A cut 30 inches deep will sever practically all of the connecting roots between trees and is believed to be sufficient to arrest the underground spread of the disease.

Two power tool methods have been successfully used to sever connecting roots between trees. Wysong has used a trenching machine. Riker devised a plow with a knife blade which makes a 30-inch deep slice in the soil without digging a trench. Each of these methods is faster than hand trenching.

The spread of the disease into a new area on transplanted infected small trees is a possibility, but information on this subject is very limited.

There is little likelihood of spreading the disease on dimensional lumber without bark or sapwood cut from infected trees. The fungus is usually confined to the recent annual rings and would be largely removed when logs are cut into lumber. We have no evidence that the oak wilt fungus can be spread from such material. The fungus has been isolated from the outer rings six months after the death of the tree, and from stumps two years after trees were killed by oak

wilt, but further investigations are necessary to determine just how long it may remain alive in dead standing or cut trees.

Need for Research

Much research has been conducted on oak wilt but a great deal remains to be done. It is of primary importance to determine how the disease spreads a considerable distance from an infected area to healthy trees. We need also to know if the fungus ever fruits externally on diseased or dead trees. This knowledge would help to provide a basis for the development of a sound and practical control.

Knowledge of how long the fungus remains viable in dead trees or in stumps, and if it spreads from such material to healthy trees, is needed to determine if sanitation work is necessary and justifiable.

More information is needed on the relative susceptibility of the different oak species. A search should be made for resistant individual trees; breeding oaks for resistance coupled with desirable growth characteristics might produce exceedingly valuable disease-resistant forest, shade and ornamental trees.

Need for Surveys

The information obtained during the past year on the distribution of oak wilt has not only revealed that the disease is far more widely dispersed than previously known, but also that we do not know just where it may be. Any consideration of local control, or of wide-scale control, or attempts to estimate the importance of the disease and forecast its future progress must be based upon a more complete knowledge of its distribution and intensity than is now available.

A survey should be made in each of the States where oaks are of commercial importance. A complete survey would not be immediately possible, but spot checks could be made with a reasonable expenditure of effort.

Flights made in low-flying airplanes in parallel lines from 10 to 50 miles apart would probably reveal the presence of oak-wilted trees if the disease were well established in a State. More intensive surveying would be justified in areas with high oak values.

One observer in a light airplane can survey about 17 or 18 square miles of forest land per hour. Two observers could survey approximately twice that area. This is based on an air speed of about 80 miles per hour and each observer surveying the forests in a 1/4 mile wide strip. About 10 to 12 percent has been deducted from the area possible to cover to compensate for the circling that must be done to examine closely a suspicious dying tree and to enable the observer to locate accurately an area on a map and relate it to conspicuous landmarks. The above estimates do not take into consideration the time spent in flying over non-forested land or from the airport to the areas to be surveyed. In 1950, in the Midwest, we found that the average rental for a small plane of the Stinson type with pilot was $15.00 per hour.

Future Plans

The Division of Forest Pathology has planned additional research on oak wilt. It also hopes to continue survey work in 1951. In all of these activities we solicit the cooperation of the various State and private agencies who have an interest in oaks. We have asked each of the States where oaks are of commercial importance to conduct surveys of their own if possible. We have agreed to coordinate the work in the different States and to disseminate information resulting from these surveys to all interested parties. Arrangements have not yet been made in each State for the culturing of specimens collected from trees suspected of having oak wilt. Unless otherwise advised it would probably be best to send such specimens to the Department of Plant Pathology of the State Agricultural Experiment Station or College in the State in which specimens are collected.

DIVISION OF FOREST PATHOLOGY, U. S. PLANT INDUSTRY STATION, BELTSVILLE, MARYLAND

OAK WILT IN KANSAS

Ivan J. Shields

Chalara quercina Henry, the fungus that causes oak wilt, has been isolated from Quercus stellata in Kansas.

Eight dead and eight dying oaks were found in a wooded pasture in Douglas County, about one mile northeast of Baldwin. The dead trees had apparently been dead for from one to several years. Fruiting bodies of a Polyporus species were present on many of the larger branches.

The dying trees exhibited symptoms of oak wilt as described by Henry et al. [1], though the dark brown streaking of the outer sapwood was not prominent. These dead and diseased oaks were all within an area of 100 feet in diameter.

The owner stated that the trees in the area had been dying for about 15 years. As trees died they had been removed and used for firewood. When asked whether the trees killed earlier had retained the foliage for some time after dying, he was not positive.

On July 13 samples of wood were taken from several diseased trees and isolations were attempted. Five colonies of the same fungus were obtained. On August 1, the cultures obtained by transfer from these colonies were examined but no spores were found. The cultures then were placed in refrigeration until October, when new transfers were made. In two weeks these showed characteristic conidiophores and endoconidia of Chalara quercina. A culture was sent to A. J. Riker, who verified the identification. [2]

During June and July, 1950, 30 counties in the eastern quarter of Kansas were visited in search of oak wilt. This area contains most of the oak timber of the State. One tree of Q. velutina showing symptoms suggesting wilt was found about one mile northeast of the locality mentioned above. No isolations were attempted. A diseased tree of Q. velutina was reported from Chanute, but the cause of the trouble has not been determined. Beyond these two possibilities no other evidence of oak wilt was found.

This is believed to be the first report of oak wilt from Kansas. As far as these observations go its present distribution in the State seems to be limited.

DEPARTMENT OF BOTANY, UNIVERSITY OF KANSAS, LAWRENCE, KANSAS

BLEEDING CANKER OF MAPLE IN NORTH CAROLINA AND TENNESSEE

E. Richard Toole

Bleeding canker, caused by Phytophthora cactorum, [1] has been found on red maple in the Cherokee National Forest in eastern Tennessee. In this area, where red maple makes up about 2 percent of the stand, cankered trees were commonly observed. Cankered trees could be distinguished for some distance by the reddish coloration of the foliage even in midsummer, and in addition, leaves were often dwarfed and yellow green. Examination of the trunks showed long irregular cankers near the base of the tree, from which sap had exuded. Bleeding canker was found on forest-grown red maple in Monroe, Johnson, Unico, and Polk Counties, Tennessee, and in Avery County, North Carolina. One ornamental sugar maple in Morganton, North Carolina, was affected with bleeding canker. This disease has previously been reported only on ornamental maples and only in the Northeast from Massachusetts, New Jersey, and Rhode Island.

DIVISION OF FOREST PATHOLOGY

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[1] Henry, B. W., C. S. Moses, C. Audrey Richards, and A. J. Riker. Oakwilt: its significance, symptoms, and cause. Phytopath. 34: 636-647. 1944.
[2] Riker, A. J. Letter of December 12, 1950 to A. J. Mix.

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[1] Fungus identified by W. A. Campbell.

BRIEF NOTES ON PLANT DISEASES

TOMATO LATE BLIGHT IN KANSAS

By Claude King and C. T. Rogerson

Late blight caused by Phytophthora infestans (Mont.) DBy. was found on tomato fruits at Concordia, Cloud County, Kansas, in November, 1950. This is the first definite record of late blight of tomatoes in Kansas.

Identification of the disease based on symptom expression on the host revealed that the disease also occurred in Douglas, Doniphan, Johnson, Shawnee and Wyandotte Counties. It was also reported from Osage County by the County agent. Probably the disease occurred generally throughout northeast Kansas. Severe losses were reported from Doniphan and Osage Counties.

The unusual amount of cool and rainy weather during the summer of 1950 in Kansas undoubtedly accounts for the development of tomato late blight. The first indications of the disease were noted in August. In normal years the warm and dry weather conditions prevalent throughout Kansas have without doubt kept the disease in check.

KANSAS STATE COLLEGE, MANHATTAN, KANSAS

LENTICEL-LIKE BLEMISHES ON TOMATO FRUITS

By O. H. Calvert and P. A. Young

Fields of tomatoes near Batesville, Texas, in October and November 1950 had fruits that showed abnormal, usually thin, raised lines of various lengths and designs. Many of the lines anastomosed and formed irregular rings, U's and T's, while others formed fan-like ends. On young fruits (1 inch in diameter) the raised lines were short and had indistinct ends. The parenchyma cells of the normal part of the epidermis became prosenchyma-like in structure in the abnormal areas. These areas were ridges with slightly brown tops. On older green fruits (2 to 3 inches in diameter) the lines were 1/2 to 1 mm. wide and consisted of lenticel-like structures separated by prosenchyma-like islands of epidermal tissue. The ends and sides intergraded with normal epidermal parenchyma. Some of the plants had many abnormal fruits while others had few or none. The corky brown-line blemishes lowered the grade of the fruits on the market. The plants showed no abnormalities of the leaves or stems. The cause of this fruit abnormality is undetermined. Five large fields of Stokesdale tomatoes showed 5 to 60 percent of the fruits with the abnormal lines. Two nearby fields of Rutgers tomatoes showed traces (less than 1 percent) of the fruits with this abnormality.

TEXAS AGRICULTURAL EXPERIMENT STATION, WINTER HAVEN AND JACKSONVILLE

ADDITIONAL OLD WORLD DISTRIBUTION OF STEM AND FOLIAGE SCAB OF SWEETPOTATO

By Lee Ling and Anna E. Jenkins

During his travels in southeastern and southwestern China, several years ago, the first writer observed a disease of stems and leaves of sweetpotato that he later recognized as stem and foliage scab, of the history given by Viégas and Jenkins. [1] To this apparently new record of the disease in the Old World is to be added another from the Caroline Islands. Following the second writer's suggestion to Dr. F. R. Fosberg that during his botanical exploration in the Caroline Islands he be on the lookout for stem and foliage scab of sweetpotato, he was successful in finding the malady on Fefan Island. The label of the specimens he contributed bears the following collection data: "Fefan Island, saddle between Mt. Chukusou and M. Chukuchap, between Mesa and Sanuk villages. Moist open slopes. Alt. 225 m., May 27, 1946. Coll. F. R. Fosberg 24506. Det. A.E.J."

The geographical range of stem and foliage scab as previously known comprised Formosa, Amami Islands, and Guam in the Old World, and Brazil in the New World. The pathogen of the disease is Elsinoë batatas Viégas & Jenkins (Sphaceloma batatas Saw.). [1]

FOOD AND AGRICULTURE ORGANIZATION OF THE UNITED NATIONS AND DIVISION OF MYCOLOGY AND DISEASE SURVEY, BUREAU OF PLANT INDUSTRY, SOILS, AND AGRICULTURAL ENGINEERING

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[1] Viégas, A. P. and A. E. Jenkins. Stem and foliage scab of sweetpotato. Jour. Agr. Res. 33: 244-249. 1943.

A REPORT TO THE CONTRIBUTORS TO OUR PLANT DISEASE REPORTER

Paul R. Miller

The following statement combines our answer to a request for the requirements that articles should meet to be accepted in the Plant Disease Reporter; with a discussion of some other topics connected with this publication.

OBJECTIVES, STANDARDS, AND FORM OF THE REPORTER: -- We believe that the Reporter's outstanding service is the prompt publication of current information on plant diseases and on new developments in plant pathology. Especially is it an outlet for tentative or unconfirmed conclusions, discoveries, predictions or suspicions that would lose much of their value as guides or warnings if not reported at once.

In addition to news, the Reporter also provides space for historical and geographical reviews, economic analysis, and discussion and speculation on various aspects of plant pathology.

Content vs. Form in Judging Acceptability of Papers: Papers are judged primarily on content. By content is meant not only the general subject matter but also the manner in which the article's specific contributions are presented. Form, i. e., arrangement of content otherwise suitable, is left to the author.

Specific Requirements for Content, Form, and Scientific Level: For content we have some guiding principles rather than specific requirements. Brief presentation of subject matter within the Reporter's field as outlined above, with enough data and analysis to allow clear understanding of the fundamental problem and of procedures, results, and conclusions, is preferable for the Reporter. Complete highly technical accounts with full details are ordinarily best published in other journals.

As said above, except for minor matters arrangement of papers is left to the author. For long papers some division into sections is helpful to the reader, and we sometimes supply headings, but we do not have a set form except for Literature Cited, in which articles are listed alphabetically by authors and numbered for reference in the text. The author's professional affiliation should be given as briefly as possible, at the end of the article. It does not seem necessary to give the exact position that a writer holds, but the institution, or institutions, with which his work is connected, and the address at which correspondents may reach him, are essential. Except in unusual cases where more specific information is necessary, we prefer not to use footnotes for this purpose.

Footnotes should be typed at the bottom of the page.

It should be remembered that no variation in type size or style is possible in the Reporter.

As to whether a paper is sufficiently scientific, we go by the fundamental objective of the Reporter. If an article contributes to any aspect of our knowledge about plant disease occurrence, etiology, history, control, or effects; if it reports an advance in methods of plant disease study; or if it stimulates thought about the potentialities and responsibilities of plant pathology as a science and a profession; then we think that it is worthy of a place in the Reporter

We do not accept articles naming new species or varieties of organisms or making new combinations in scientific names; this is mainly to avoid complicating taxonomic literature.

Editorial Review: Review in the first place is to determine acceptability for the Reporter. Clearly unsuitable papers are returned at once. Where there is doubt the paper is passed on to one or more colleagues for critical reading. Some doubtful articles are accepted as having an unusual but definite connection with plant pathology. Others that may be suitable as to subject but unacceptable as to presentation are returned with suggestions for restyling, or an alternate place of publication may be suggested. Except for matters involving policy, which are referred to the Chief of our Bureau, the Survey is the final judge of the acceptability of a manuscript for the Reporter. In general, pathologists understand the functions and special values of the Reporter very well, so that manuscripts submitted are seldom questioned.

After a manuscript has been accepted further review is merely to prepare it for typing. Obvious errors are corrected and sentences may be reworded slightly. Except in unusual cases, however, we will not do any actual rewriting. Accuracy and style, we believe, are the author's responsibility. Nor do we check literature references.

We are careful to check the correctness and spelling of scientific names of organisms. If the nomenclature differs from that followed by us, we insert our own preference in parentheses for uniformity in reference and indexing.

Perhaps I might make one suggestion. In purely pathological articles it does not seem necessary to give the authority for the scientific name of an organism, except when omission would be likely to lead to confusion as to the identity of the organism involved.

To obtain complete understanding from readers, we think it essential to state the scientific names of causal organisms, since disease names are often indefinite, or differently used in different regions. If scientific names are omitted we often insert them, when we are certain that we know what they should be. However, to avoid the possibility of error or misunderstanding, we much prefer that authors should supply these names themselves.

NAMES OF CHEMICAL CONTROL MATERIALS: -- Use a trade name only to designate the specific product of the company to which the name belongs. To avoid misunderstanding spell the name exactly as the manufacturer does. If the chemical composition is known, state it in parentheses.

Use the coined common names for chemical compounds when available. These names should be spelt with initial small letter, whereas proprietary names should begin with capitals.

TABLES AND ILLUSTRATIONS FOR THE REPORTER: -- Tables often give us trouble in setting up the Reporter. Large or complicated tables are difficult for us to reproduce. Also, titles and headings given to tables by authors are often indefinite, or are not self-explanatory as they should be. Tables should be carefully thought out, with particular attention to our limitations in reproduction, and should always be checked with the text of the article.

We can handle illustrations best when they are sent to us unmounted. Be sure to pack them carefully to prevent injury in transit. (Although it might seem that mounting should be a protection, we have received some mounted photographs that were badly bent, and in such cases it is very difficult to repair the injury). One method used by some contributors is to put all the prints for the illustrations in an envelope between sheets of heavy rigid cardboard, marking each print lightly on the back with its proper figure number and designating top and bottom if it is not obvious. Legends for the illustrations are then typed separately on manuscript sheets.

Remember that, in general, illustrations undergo the same 25 percent reduction as the text and allow for it in the scale of charts or pictures, as well as in printed or typed legends, so that the final reproduction will not be too small to show the desired detail or the legends too small or faint to be legible. On the other hand, illustrations that are too large are hard to fit into the text and sometimes require special handling. The over-all dimensions of our typed page, exclusive of heading, are 8 by 11 1/4 inches. Please keep this in mind when planning for illustrations, and tables as well.

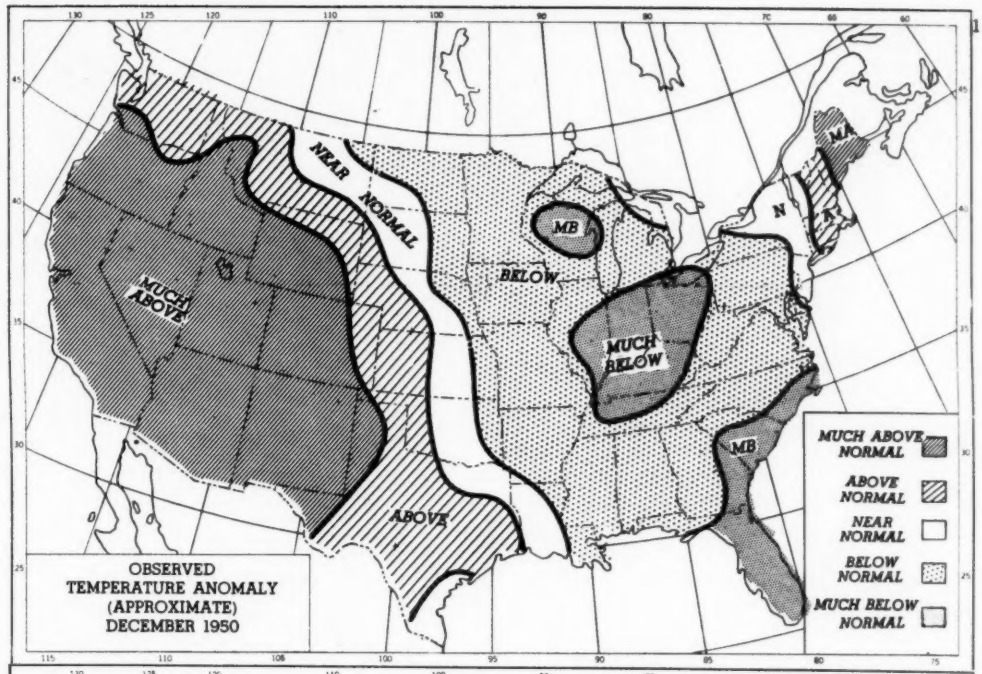
INCREASING COSTS BRING CONSEQUENCES: -- In the Survey we are very proud of the Reporter. I am sure, however, that the problems we face in publishing it are not very generally appreciated. Its prestige had already increased greatly in the last few years because of the type of contents and the promptness of publication, and since our recent change to the new method of reproduction its popularity among plant pathologists has increased by leaps and bounds. Thus, increased demand for space in the Reporter, plus increased expense per page, plus a great increase in work for a non-increased staff, plus the fact that the journal is paid for out of our appropriation and we receive no compensation for subscriptions to it -- with all of these acting together our problems as regards the Reporter are approaching the acute stage. Moreover, in the foreseeable future, conditions are not likely to become easier.

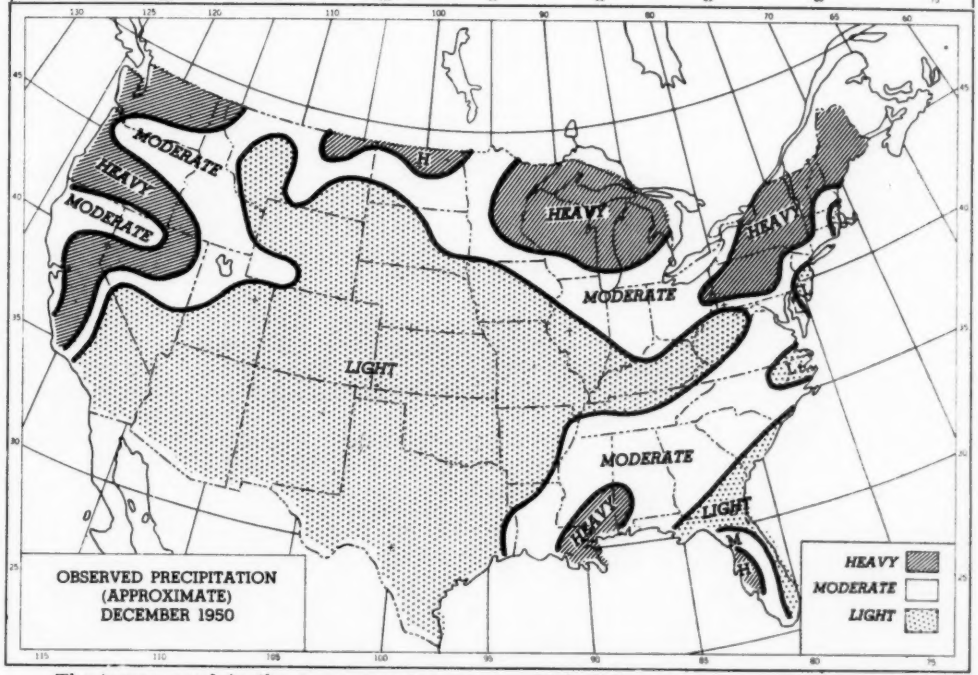
Therefore, we ask authors to present essentials as clearly and briefly as possible. There is no need to sacrifice readability and clarity for the sake of brevity, however. We will attempt to handle any work in the Reporter's field that really needs to be published.

Reprints Can No Longer Be Furnished: One step we are forced to take, however regretfully. In the past we have furnished requested reprints of articles appearing in the Reporter, free of cost, since there is simply no method by which we could be reimbursed. As of now we will no longer be able to supply them.

Multilith Plates Available: However, if they can be used, we will be glad to send the multilith plates to the author or station, when requested to do so at the time the manuscript is submitted. The press size of these plates used for the Reporter is designated as small -- maximum image 9 1/2 by 13 inches, maximum paper size 9 3/4 by 14 inches -- which is Model 1250. I believe most of the Agricultural Experiment Stations have this type of multilith machine.

DIVISION OF MYCOLOGY AND DISEASE SURVEY





The terms used in the accompanying maps, which define the ranges of temperature and precipitation, are numerical class limits. These are based on a statistical analysis of past records through which is determined the normal frequency of occurrence of temperatures and precipitation at various times of the year for different locations. For temperature the classes above, below, and near normal are so defined that they each normally occur one-fourth of the time; much above and much below normal, one-eighth of the time. Precipitation is depicted in terms of light, moderate, and heavy, each class normally occurring one-third of the time and thereby having equal probability of occurrence. These maps graphically represent only the general trends and give the country's weather picture at a glance. For quantitative studies, where monthly mean temperatures and actual precipitation records are needed for a given time and place, other publications of the Weather Bureau should be consulted. P. R. M.